A PROSPECTIVE STUDY OF EVALUATION OF PERI-OPERATIVE INTRA-ABDOMINAL PRESSURE MONITORING AND ITS ASSOCIATION WITH POST-OPERATIVE MORBIDITY AND MORTALITY IN EMERGENCY LAPAROTOMY

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BONAFIDE CERTIFICATE

This is to certify that dissertation named **"A PROSPECTIVE STUDY OF EVALUATION OF PERI-OPERATIVE INTRA-ABDOMINAL PRESSURE MONITORING AND ITS ASSOCIATION WITH POST-OPERATIVE MORBIDITY AND MORTALITY IN EMERGENCY LAPAROTOMY"** is a bonafide work performed by Dr.S.Maithreyi, post graduate student, Department of General Surgery, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment of regulations of the Tamilnadu Dr. M.G.R Medical University for the award of M.D. Degree Branch I (General Surgery) during the academic period from May 2011to April 2014.

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This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.S. Branch I (General Surgery)**.

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ABSTRACT

BACKGROUND AND OBJECTIVES

Abdominal compartment syndrome (ACS) and intra-abdominal hypertension (IAH) have been prominently identified among patients with surgical emergencies such as pancreatitis and intestinal obstruction, and among patients in Intensive Care Units (ICU). It is a proven fact that IAH is rampant among ICU patients. Around 18 to 80% ICU patients are affected by this entity and hence the increasing interest in this topic. Most of the body systems are affected by ACS and IAH, most markedly the renal, respiratory, cardiac and nervous systems. The prognosis of the patient depends on the flow of the blood to various organs which are ultimately affected by ACS/IAH. Timely recognition and appropriate treatment of ACS/IAH, either medical or surgical, plays a very important role in reducing the morbidity and mortality of patients. The aim of the study was to assess the relationship between peri-operative intra abdominal pressure and post-operative patient morbidity, (with special reference to kidney function and pulmonary atelectasis) , and mortality.

METHOD

Two groups of 50 surgical patients were selected. The experimental group had increased intra-abdominal pressure. The control group were selected from those undergoing elective surgery. In both groups, patients with factors which could cause increased intra-abdominal pressure like obesity were excluded.

The preliminary details collected from patients included, name, age, sex, diagnosis, operative procedure planned, BMI. Presence or absence of diabetes mellitus, hypertension. Examination findings of pulse, blood pressure, respiratory rate, temperature and specific systemic examination of respiratory system, cardiovascular system and abdomen were noted.

Laboratory tests done preoperatively and post operatively according to need were hemoglobin concentration, plasma urea, plasma creatinine and Chest X-Ray. Intra-abdominal pressure and urine output were monitored pre-operatively and at four hourly intervals in the post-operative period for 24 hours.

The intravesical route of measuring the IAP was using the Foley's method. Measurements were made at regular intervals, usually varying from 4 to 6hrs. Depending on the IAP the treatment modality was adjusted.

Operative findings during laparotomy and the surgical procedure done were noted. Post-operative mechanical ventilation and re-laparotomy were followed

up if needed. Also, cause of mortality was noted if the patient expired in spite of maximal supportive medical care.

RESULTS

The results were documented and tabulated. The association of intra-abdominal pressure with renal dysfunction and pulmonary atelectasis was studied. The statistical significance was found out using the Chi square test.

INTTEPRETATIONS AND CONCLUSION

It was found that the incidence of increased intra-abdominal pressure (emergency) was more among men. Urea and creatinine were significantly raised in the increased intra-abdominal pressure group when compared to the normal intra-abdominal pressure group at all time intervals (pre-operative, post – operative, 4^{th} hour, 12^{th} hour and 24^{th} hour), thus indicating renal dysfunction in cases of increased intra abdominal pressure. There was no significant correlation between the urine output and the inra-abdominal pressure.

There is a mild increase in the incidence of atelectasis post-operatively in cases of increased intra-abdominal pressure as compared to the group with normal intra abdominal pressure. The mortality was 0% in GRH.

KEYWORKS

Intra-abdominal pressure, intra-abdominal hypertension, abdominal compartment syndrome, renal dysfunction, atelectasis, urea, creatinine, urine output

INTRODUCTION

Abdominal compartment syndrome (ACS) and intra-abdominal hypertension (IAH) have been prominently identified among patients with surgical emergencies such as pancreatitis and intestinal obstruction, and among patients in Intensive Care Units (ICU). It is a proven fact that IAH is rampant among ICU patients. Around 18 to 80% ICU patients are affected by this entity and hence the increasing interest in this topic. Most of the body systems are affected by ACS and IAH, most markedly the renal, respiratory, cardiac and nervous systems. The prognosis of the patient depends on the flow of the blood to various organs which are ultimately affected by ACS/IAH. Timely recognition of ACS/IAH, its clinical features and risk factors play a very important role in reducing the morbidity and mortality of patients. It becomes essential to have a sound knowledge of the pathophysiology to identify and plan an appropriate therapeutic approach.

Compartment syndrome is a well known phenomenon in the extremities. Increased pressure in closed fascial spaces of limbs causes a decrease in the perfusion pressure. When perfusion is depressed beyond a critical level, tissue viability is lost. The same principle applies to the intra-abdominal space which is also a closed compartment. The normal Intra-abdominal pressure is <7mmHg. Various conditions which ultimately lead to the accumulation of fluid, flatus or feces increase the intra-abdominal pressure and lead to intra-

abdominal hypertension first and later abdominal compartment syndrome. Injuries and diseases affecting the abdomino-pelvic region such as intestinal obstruction and peritonitis cause primary abdominal compartment syndrome; whereas diseases originating outside the abdomen such as sepsis and major burns leads to secondary abdominal compartment syndrome.

There are no specific radiological features to identify abdominal compartment syndrome. This is one reason why CT scan is not used to diagnose a case of Intra-abdominal hypertension. However, radiological investigations may help to identify the cause, severity and the potential complications of the causative illness for the increased pressure. Whatever the cause is, rapid identification and diagnosis of intra-abdominal hypertension can be done using a simple bedside test, the intra-vesical pressure measurement.

Several studies have shown that the incidence of IAH and ACS is significantly more when associated with sepsis and septic shock; it may be as high as 85% and 30% respectively. In cases of pancreatitis about 40-70% patients have been recognized to have IAH and 10-50% to have ACS. Post laparotomy incidence is variable but is generally low with elective surgeries and higher following emergency procedures.

The World Society on Abdominal Compartment Syndrome (WSACS) was established in 2006 and came to be the ultimate authority on IAH/ ACS.

REVIEW OF LITERATURE

Richard Volkmann in 1811 was the first person to describe compartment syndrome. He described it with regard to the fascial spaces of the limbs. He published an article entitled "Die ischemischen Muskellahmungen und – Kontrakturen"[2]. He had observed that increased pressure within a closed fascial space reduced the perfusion of the muscles within and finally led to contracture.

Etienne-Jules Marey in 1863, was the first to describe the relationship between respiratory function and increased IAP. He published a paper entitled "Physiologie medicale de la circulation du sang" where he observed an inverse relationship between the respiratory effort and perfusion of abdominal viscera. Paul Bert in 1870, reinforced Marey's conclusion. He published the book "Lecons sur la physiologie de la respiration" in which he described the descent of the diaphragm and elevation of IAP on inspiration, based on animal models. He measured the abdominal and thoracic pressures with tubes in the rectum and trachea respectively [2].

There were numerous authors who experimented on the measurement of IAP and the best to monitor it was Schatz, a German physician. In 1872, he used a balloon tube connected to manometer and measured the pressure within the uterus [2]. One year later, another German, Wendt, measured the IAP through

the rectum. And later in 1875, Oderbrecht made a similar measurement using a catheter inserted via the urinary bladder.[2]

Mr.E.Emerson in 1911 conducted several experiments in dogs and concluded that the IAP increased on contraction of the diaphragm, while anesthesia and muscle paralysis caused a decrease in the IAP. He also quoted that an increased IAP may cause death as a result of cardiac failure. His most significant observation was that cardiovascular collapse can be due to "distension of the abdomen with fluid or gas, as in peritonitis, ascitis or typhoid fever" and that "relief of the strained heart is always seen after removal of the excess fluid (ascitis)"[2]. The credit for building the foundation of the clinical and experimental research on IAP in the 20th century itself goes to Emerson.

Few decades elapsed after Emerson's significant observations without any progress or research in the field. Mr.W.H.Ogilvie in 1940, published an important article in the Lancet regarding the better prognosis of the patients, by keeping the abdomen open after war wounds [2]. This was reinforced by Mr.R.E.Gross in 1948 who acknowledged the importance of avoiding tight abdominal closure under excessive tension [2]. In1951, M.G.Baggot noted an interesting finding in cases of increased IAP. He identified that a main factor increasing IAP was abdominal dehiscence and strongly advised against tight closure of the abdomen under tension and recommended leaving the abdomen open[3].

In 1984, I.Kron, P.K.Harman and S.P.Nolan were the first to describe the entity ACS. They also described a simple and reliable diagnostic technique of placing an indwelling transurethral bladder catheter for this purpose. In post laparotomy patients, in the absence of renal insufficiency or rapid blood loss, an IAP above 20 mmHg was an indicator for continued and vigilant observation. In post operative patients, an IAP of above 25mmHg with low urinary output and adequate blood volume was described as an indication for re-exploration and abdominal decompression [4].

The term abdominal compartment syndrome was first introduced in 1989, by Fietsam et al, in four patients who had increased intra abdominal pressure following repair of ruptured aortic aneurysm. It was recognized by increased central venous pressure, decreased urinary output and increased ventilatory pressure associated with massive abdominal distension in the absence of bleeding. These set of findings represent the abdominal compartment syndrome caused by massive retroperitoneal and interstitial swelling.[2]

In 2004, the World Society of Abdominal Compartment Syndrome (WSACS) was established by an international group of surgeons and physicians[5]. They recognized the need for a cohesive approach fostering education, promoting research, treating and improving the survival of patients suffering from IAH and ACS. They achieved a great deal within three years of their establishment, developing definitions and recommendations based on

international consensus and evidence. They established a worldwide network of research scientists and clinicians, published their first textbook on ACS, filled with the latest research findings [5]. The WSACS currently serves as a scientific resource and a forum for establishing the concept of IAH and ACS in everyday clinical practice [1,6].

Several guidelines and algorithms set by the WSACS have been used widely and have significantly brought down the mortality rate and improved the survival of patients suffering from IAH and ACS.

Definitions, description of ACS and measurement techniques approved by WSACS

Compartment syndrome occurs when there is increased pressure within a closed anatomical space, which causes decreased tissue perfusion and hence threatens the viability of concerned tissues. IAH affects diverse systems and organs, especially renal, cardiac, respiratory and nervous systems. Function of these systems can be easily evaluated and monitored in the critical care unit. Decrease in tissue perfusion is associated with increased afterload, decreased preload and extrinsic compression leading to hypoperfusion and decreased end organ oxygen delivery. This phenomenon when occurring as a result of pressure-volume deregulation in the abdomen is termed Abdominal

Compartment Syndrome. Thus ACS is not a disease but a syndrome with a group of specific sign and symptoms.

Definitions

1. Intra-abdominal Pressure

The abdominal compartment is a closed cavity with fixed rigid and flexible walls. The fixed walls are the pelvis, spine and the costal arch; the flexible walls being diaphragm and the abdominal wall. The pressure within the abdomen at any given time is determined by the elasticity of the flexible wall and the character of the abdominal contents [1]. The contents of the abdomen are primarily fluid in character and relatively non-compressible. In accordance to Pascal's law, the pressure measured at any point in the abdominal cavity represents the IAP throughout the abdomen [2]. Hence intra abdominal pressure is considered as a steady state pressure present within the abdominal cavity. IAP decreases with diaphragmatic relaxation during expiration and increases with diaphragmatic contraction at inspiration [2]. Also, it is directly affected by the volume of the hollow viscera (which may be filled with fecal matter, liquid or air) and the solid organs, the presence of blood, ascitic fluid or other space – occupying lesions (gravid uterus or tumor) and conditions such as third space edema and burns which limit the expansion of the abdominal wall.

The normal IAP ranges upto 7 mmHg. Few physiological conditions such as pregnancy and morbid obesity are associated with chronically elevated IAP. Change in body position, mechanical ventilation, sepsis, organ failure and recent abdominal surgery are associated with increased IAP. [8]

2. Abdominal Perfusion Pressure

Mean arterial pressure (MAP) minus IAP is Abdominal Perfusion Pressure (APP). The main factors influencing MAP and IAP are arterial inflow and the resistance to venous outflow respectively. APP is a reliable predictor of visceral perfusion and a possible endpoint for resuscitation. Therefore APP could be a superior parameter in predicting patient survival in cases of IAH and ACS [17]. An APP greater than 60 mmHg has been associated with better patient prognosis [9].

3. Filtration Gradient

The difference between the glomerular filtration pressure (GFP) and proximal tubular pressure (PTP), which is the mechanical force promoting filtration across the glomerulus is known as the renal filtration gradient (FG). PTP is assumed to be equal to IAP in cases of IAH.

GFP = MAP -2 x IAP [9]

Hence changes in IAP will affect renal filtration and urine production rather than MAP. Oliguria is one of the first sign of increased IAH [9,10].

4. Intra-abdominal hypertension (IAH)

In healthy adults, the normal IAP is <7 mmHg [11]. In conditions such as morbid obesity, chronic obstructive pulmonary disease and pregnancy, the physiological upper limit is accepted to be 12mmHg by the WSACS [5]. This reflects elevated normal pressure from conditions that exert external pressure to the diaphragm or the peritoneal envelope.

IAH is the sustained or repeated pathological increase of the intra abdominal pressure above 12 mmHg [12].

Grades of IAH according to the level of IAP [14]

Grade I : 12 – 15mmHg
Grade II : 16 – 20mmHg
Grade III : 21 - 25mmHg
Grade IV : >25mmHg

Subclassification of IAH according to duration [2]

Hyperacute : Elevation of IAP for a few seconds to minutes as in straining, sneezing, defecation, laughing, coughing

Acute : Elevated IAP present over a few hours, seen in surgical cases (e.g. intra abdominal hemorrhage or trauma)

Subacute : Elevated IAP present over days and found in medical cases

Chronic : IAP elevation develops over months (E.g. pregnancy) or years (E.g. Intra-abdominal tumour, chronic ascitis, morbid obesity, cirrhosis, peritoneal dialysis). These patients may develop either acute or subacute IAH when severely ill

5. Abdominal Compartment Syndrome (ACS)

In the majority of patients, Critical IAP seems to be between 10 and 15mmHg. There is microcirculatory reduction in the blood flow at this pressure and it indicates the initiation of ACS. IAH progresses to ACS as end organ dysfunction develops [12].

ACS consists of the following triad [9]

a. Pathological acute increase in IAP >20-25mmHg
b. Adverse effects on end-organ function
c. Beneficial effects as a result of abdominal decompression

ABDOMINAL COMPARTMENT SYNDROME - CLASSIFICATION

According to the cause and duration, ACS may be classified as Primary, secondary or recurrent[9]

1. Primary ACS (abdominal or surgical ACS) – Presence of acute or subacute IAH which results from intra abdominal cause. (post-abdominal surgery or abdominal trauma)
2. Secondary ACS (extra-abdominal or medical) Presence of subacute or chronic IAH from conditions that require massive fluid resuscitation, e.g. major burns or septic shock
3. Tertiary or recurrent ACS – Presents with resurgence of ACS following resolution of a prior episode

ABDOMINAL COMPARTMENT SYNDROME – RISK FACTORS [2]

1. Decreased abdominal wall compliance
    a. Abdominal surgery with tight primary closure – gastroschisis, large hernias
    b. Acute respiratory failure- especially with increased intra thoracic pressure

c. Mechanical ventilation, especially patient-ventilator dyssynchrony , use of accessory muscles of respiration
d. Increased positive end expiratory pressure
e. Positioning – prone, head end elevation >30 degree
f. Basal pneumonia
g. Central obesity, high BMI
h. Major burns/ trauma

2. Increased abdominal contents

a. Liver dysfunction / ascitis
b. Haemoperitonium/ pneumoperitoneum
c. Peritoneal dialysis
d. Major trauma

3. Capillary leak/fluid resuscitation

a. Hypotension
b. Acidosis
c. Polytransfusion (>10 units blood/24hrs)
d. Hypothermia (core temp <33c)
e. Coagulopathy (prothrombin >15s, international standardized ratio >1.5, platelet <55,000mm3, partial thromboplastin time >2times)

f. Oliguria

g. Pancreatitis

h. Major burns/trauma

i. Sepsis

j. Damage control laparotomy

4. Post-operative

a. Edema following extensive dissection

b. Reduction of diaphragmatic hernia

c. Laparoscopic surgery with insufflation of intra-abdominal air

d. Ileus

e. Damage control laparotomy

f. Visceral edema post fluid resuscitation

g. Polytransfusion

h. Intra or retroperitoneal bleed

i. Peritonitis or intra abdominal abscess

ABDOMINAL COMPARTMENT SYNDROME – CAUSATION

In critically ill patients, massive fluid resuscitation could be a major cause for the development of ACS. In patients with increased vascular permeability due to inflammatory response syndrome, massive fluid resuscitation leads to fluid sequestration and ascitis formation which leads to increase IAP. During the resuscitation of patients with large amount of fluids, bowel edema with ingurgitation of mesenteric vessels and lymphatics occurs. Fluid sequestration into the peritoneal cavity causes increased IAP which leads to IAH. IAH decreases the venous return, thereby creating a vicious cycle .

Fig 1: Duodenal Perforation with peritonitis

Fig 2 : Circumferential burns - abdomen

Burns by several mechanisms can lead to ACS. Circumferential burns of the abdominal wall lead to eschar formation and edema. This produces an extrinsic compression of the abdominal cavity raising the IAP. Large burns can cause elevated mesenteric vascular resistance which is ascribed to the massive release of vasoactive substances such as vasopressin and angiotensin II and inflammatory mediators from burned tissue. This leads to ischemic enterocolitis and bowel edema.

Massive fluid resuscitation can secondarily lead to ascitis and bowel edema which increases the IAP. All these are aggravated by the exaggerated generalised increase in capillary permeability [2].

The duration of IAH is of greater prognostic value than the absolute increase in IAP. Certain pre-existing co-morbidities such as pulmonary disease, chronic renal failure or cardiomyopathy aggravate the effects of elevated IAP, thereby reducing the threshold of IAH which leads to the clinical manifestation of ACS. In critically ill patients admitted to the ICU, screening for the IAP was recommended by WSACS [5].

ABDOMINAL COMPARTMENT SYNDROME - CLINICAL PRESENTATION

Abdominal compartment syndrome presents with varied complaints but abdominal distention and pain are common symptoms. Hypercarbia, hypoxia and oliguria are found prevalently in these patients indicating inadequate renal and respiratory functions. The sensitivity of physical examination for diagnosis and assessment is low, around 40 to 60%. Timely intervention is crucial in the management of these cases, regardless of the cause; else it would progress to multi-organ failure and ultimately death.

TECHNIQUES TO MEASURE INTRA-ABDOMINAL PRESSURE

Several techniques were proposed for the measurement of IAP and thereby the diagnosis of ACS. Clinical examination was found to be highly unreliable with a positive predictive value and sensitivity of 40 to 60%, hence making it an undependable diagnostic tool [14]. Abdominal perimeter measurements are equally inaccurate. The use of radiological investigations such as abdominal ultrasound, X-Ray abdomen, X-ray chest or computerised tomography (CT) are also erroneous in the quantification of IAP and the diagnosis of IAH. These investigations may however be used in the identification of the cause of IAH

such as abscess or hematoma and may help in deciding the management modality (drainage of collections or paracentesis)[2].

Frequent and accurate measurement of IAP is important for the identification and diagnosis of IAH/ACS. Monitoring the IAP is an accurate, safe and cost-effective method for determining the presence of IAH and deciding on the treatment protocol [15]. Given the exceedingly favourable risk benefit ratio of "measurement and monitoring IAP vs the associated morbidity and mortality of ACS/IAH", certain recommendations have been laid down.

(1) A baseline IAP measurement should be obtained if two or more risk factors for IAH/ACS are present.
(2) If the patient is identified to have IAH, serial measurements of IAP should be made throughout the course of the patient's critical illness.

IAP can be measured intermittently or continuously, either directly or indirectly. An intra-peritoneal catheter installed during peritoneal dialysis, ascitis drainage or during laparoscopic surgery can be used for obtaining the IAP directly [2].

Several methods such as intravescical, rectal, gastric, uterine, inferior vena cava and airway pressure measurements are indirect methods for obtaining IAP [16]. The intravesical route is considered the gold standard for IAP measurement and monitoring because of its simplicity and low cost [5]. This

technique is based on the fact that the wall of the bladder is very compliant. When a small amount of saline is infused, it functions as a passive reservoir and a transducer of the intra abdominal pressure. Changes in the intravesicular pressure reflect changes in the IAP sufficiently accurately for practical purposes[2]. It is important to measure the IAP with the patient in the supine position as posture affects the IAP [17].

In patients with neurogenic bladder, bladder trauma, tense pelvic hematomas and outflow obstruction, the measurement of bladder pressure is not feasible. Hence an alternate method of measurement is used to monitor the IAP via the nasogastric route [2]. The advantages of measurement of IAP via the stomach is,

(1) The problems associated with the formation of hydrostatic column of fluid in the bladder are avoided.
(2) It is easier and better for continuous monitoring of the IAP.

The intravesical route of measuring the IAP is performed by connecting the Foley's catheter to a three way tap which is then connected to a pressure transducer [5]. The patient is placed in the supine position, he is catheterised using a Foley's catheter and the residual urine is drained. Later the Foleys catheter is clamped at a point distal to the point of pressure measurement. For every 20 degree head-up tilt, the IAP increases by 2mm. The catheter is

connected to a pressure transducer and the point of mid-axillary line at the iliac crest is taken as the reference point where the intra-abdominal pressure value is zeroed. Around 25ml (if weight <20kgs, 1ml per kg) of saline is instilled into the bladder, 30 to 60secs later the reading is taken, providing time for detrusor muscle relaxation. Moreover the measurement should be taken in the absence of active abdominal muscle contraction and at the end of expiration. Measurements are taken at regular intervals, usually varying from 4 to 6hrs. Depending on the IAP the treatment modality is adjusted. As the Foleys catheter has to be clamped before each measurement, continuous monitoring of IAP using the intravesical route is challenging.

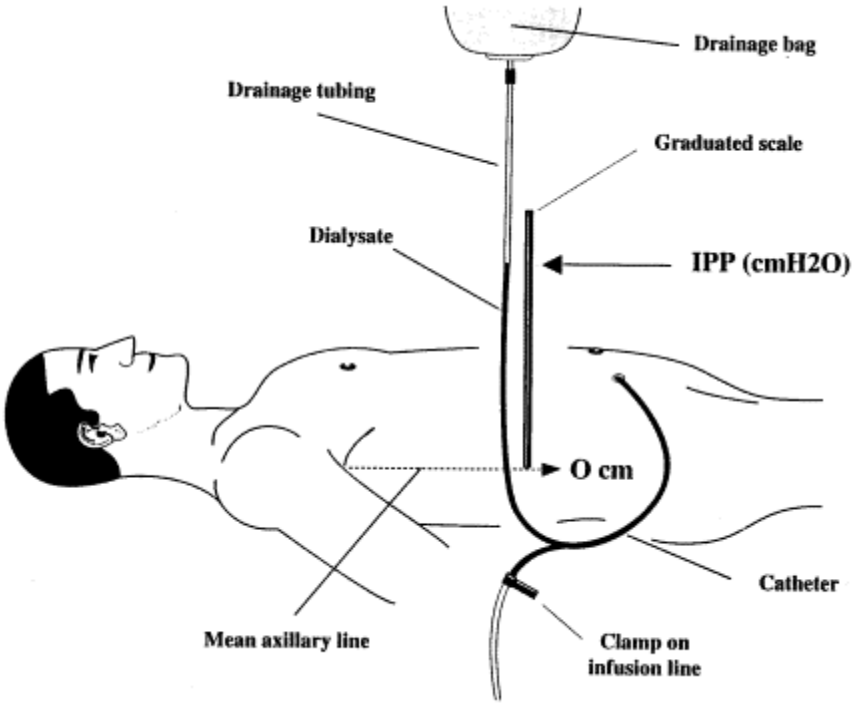


Fig 3: Technique of IAP monitoring via intra-vesical method

PATHOPHYSIOLOGY OF INTRA-ABDOMINAL HYPERTENSION/ ABDOMINAL COMPARTMENT SYNDROME

The increase in IAP adversely affects the organs in the peritoneal cavity as well as those outside the abdomen. IAH and ACS mainly affect the regional blood flow. The effects are graded and increase progressively from IAH to ACS; it is not a "all or nothing" response[1].

According to WSACS, the recognition of IAH as an independent prognostic factor for critically ill patients[15] will be gradually embedded in the "goal-directed" approach used in the ICU and will alter the decision-making process.

INVOLVEMENT OF OTHER ORGANS AND SYSTEMS

The introduction of laparoscopic surgery in the 1990s was followed by extensive experimental and clinical study of IAH and ACS and led to an increased appreciation of their pathophysiologic sequelae.[18,19] These effects include the directly affected intra-abdominal organs, as well as indirectly involved adjacent or remote systems and organs.

CARDIOVASCULAR SYSTEM

An increase in IAP causes upward displacement of the diaphragm. This decreases the intra-thoracic volume and thereby increases the intra-thoracic

pressure (ITP). This is termed abdomino-thoracic transmission, and was seen in 20 to 80% of patients. ITP is generally assumed to be half of IAP. The increase in ITP compresses the heart directly, simultaneously reducing the ventricular contractility and compliance and significantly reducing the venous return resulting in deceased cardiac output. There is an increase in the afterload / systemic vascular resistance due to compression of the aorta, pulmonary and systemic vasculature and concurrent activation of the renin-angiotensin-aldosterone pathway[20]. This causes shunting of blood away from the abdominal cavity and leads to a temporary rise of MAP which later normalises or even decreases[2]. These effects occur with an IAP of 10mmHg in a normo-volemic patient; and at a lower IAP in a hypovolemic patient[20]. Volume correction increases the preload temporarily, thereby improving the hemodynamics. It is also found that the application of positive end expiratory pressure (PEEP) aggravates the effects seen in the cardiovascular system[2,21].

The traditional intra-cardiac filling pressures such as Pulmonary Artery Occlusion Pressure (PAOP) and central venous pressure (CVP) are erroneously increased in IAH due to abdomino-thoracic transmission of pressure. Hence these parameters cannot be used for monitoring the cardiac status of the patient. Both these cardiac parameters are the sum of ITP and intravascular pressure and not reflective of the true intra-vascular volume. Thus it becomes more accurate to use volumetric indices such as global diastolic volume and right ventricular

end diastolic volume index[2]. Intravenous fluid resuscitation of the volume load and the preload responsiveness is assessed by dynamic paramaters such as stroke volume and pulse pressure[2]. If dynamic or volume parameters are not available, hemodynamic monitoring is done using traditional filling pressures[2,24].

Transmural pressure is calculated by deleting ITP which is IAP/2.

Transmural CVP = CVP – IAP/2[23]

Transmural PAOP = PAOP – IAP/2[23]

IAH causes a rise in the inferior vena caval pressure due to compression and reduced emptying, leading to a parallel rise in femoral venous pressure[2,23]. Correction of IAP restores the normal blood flow in femoral vessels. But several cases of pulmonary embolism have been reported following this normalization. This resembles the findings in ischemia-reperfusion models[2].

PULMONARY SYSTEM

The increase in the IAP causes an upward displacement of the diaphragm and increases the intra-abdominal volume[2]. This diaphragmatic displacement causes an extrinsic compression of the pulmonary parenchyma leading to atelectasis. This leads to reduced diffusion of oxygen and ventilation perfusion

imbalamce. Reduced capillary blood flow, increased alveolar dead space, decreased carbon dioxide excretion leading to hypercarbia and arterial hypoxemia occur. Both mean airway pressure and inspiratory pressures are significantly increased, while pulmonary compliance and tidal volume are reduced[2]. Changes in the ventilatory settings required to treat this secondary acute respiratory distress syndrome include

(1) Maintenance of transmural plateau pressure under 35cm of water[26]

(2) PEEP adjusted to counteract IAP

(3) extravascular lung water index to be measured due to the risk of lung edema.

URINARY SYSTEM

In patients who originally had normovolemia and normal renal functions, IAH induced renal dysfunction becomes evident as oliguria at an IAP of 15mmHg and as anuria at an IAP of 30mmHg[2]. Compression of renal vein and parenchyma and reduced renal perfusion, lead to reduced microcirculation to the functioning glomeruli and cortex. This results in tubular and glomerular dysfunction and substantially reduced urine output[10,27]. Plasma antidiuretic hormone, renin and aldosterone levels are significantly elevated[2,27]. The difference between Glomerular Filtration Pressure (GFP) and Proximal Tubular

Pressure (PTP) is the mechanical force across the glomerulus, the Filtration Gradient (FG)[30]. Renal perfusion pressure is equal to GFP and is calculated by deducting IAP from MAP. PTP is equal to IAP. Hence,

FG = GFP – PTP = (MAP-IAP) – IAP[30]

Therefore FG = MAP – 2 x IAP

Thus the IAH induced renal dysfunction and prerenal azotemia will neither be responsive to fluid resuscitation nor vasopressors such as dopamine or loop diruetics. It improves dramatically by appropriately and promptly reducing the elevated IAP[2,26].

Urinary bladder is also affected by increased IAP. Experimentally elevated IAP was found to induce structural (damage to epithelium, lamina propria and serosa), biochemical (malondialdehyde levels are increased) and contractility (bladder contraction potentiated by acetylcholine) changes in the urinary bladder[2].

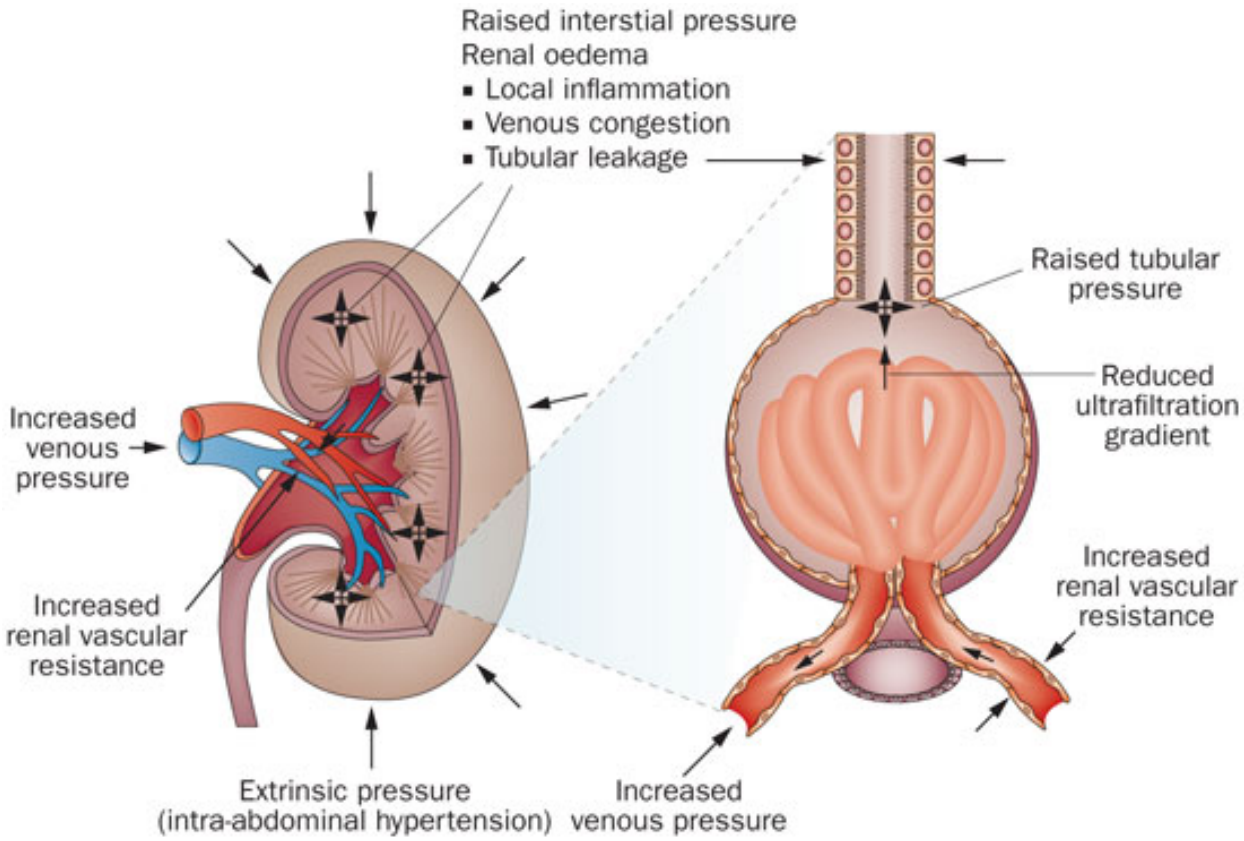


Fig 4: Pressure changes in renal system

GASTROINTESTINAL SYSTEM

The gastrointestinal system seems to be affected by even minimal change in the IAP.

a. The mucosal barrier function (affecting both bacterial translocation and influencing intermucosal nutritional flow)
b. Gastrointestinal motility are the two main functions altered.

It has been observed that the gut mucosa is very sensitive to increase in IAP. It causes

1. Compression of the mesenteric veins. This subsequently causes interstitial edema and ischemia
2. Reduction of the mesenteric blood flow, even at IAP of only 10 mmHg[2]
3. Except adrenals (due to catecholamine release), diminished blood flow to all abdominal organs[31]
4. Bacterial translocation, sepsis leading to multi-organ failure[28]
5. Decreased intramucosal pH and perfusion, increased mucosal permeability and loss of intestinal mucosal barrier function[2,32].

After repeated episodes of such insults, IAH induced ischemia-reperfusion insults, the second hit in the multiorgan failure two-hit model takes place. These effectS are called as acute intestinal distress syndrome and acute bowel injury[2]. The parameter to be monitored and maintained is to keep APP above 60 mmHg[9].

Regarding the gastrointestinal motility, a decrease in the electrical and the mechanical motor activity of the small bowel has been attributed to the increased IAP[2]. The contractile response is also inhibited by the elevated IAP.

HEPATOBILIARY SYSTEM

Even a small elevation of the IAP of around 10mmHg is associated with a reduction in the hepatic artery, vein and the portal circulation. This results in compensatory gastroesophageal collateral blood flow to the azygos vein. The liver is found to be highly susceptible to injury during IAH. Elevated IAP leads to enhanced hepatocyte proliferation, increased hepatocyte apoptosis, suggesting a liver repair response[2]. Additionally, altered glucose metabolism and mitochondrial function and deceased lactate clearance are the physiologic effects of IAH[33]. Certain conditions such as decompensated chronic liver disease, liver failure and liver transplantation are further complicated by the increase in the IAP[34].

NERVOUS SYSTEM

There have been several studies showing the concomitant increase in intracranial pressure(ICP) following increase in IAP as a part of poly-compartment syndrome[21,35].

Mechanism suggested:

1. Increased IAP causes increase in ITP which in turn increases the jugular venous pressure. This causes functional obstruction, impeding the

cerebral venous outflow, increasing the cerebral blood volume causing elevation of ICP[21].

2. Functional obstruction causing decreased lumbar venous plexus blood flow due to increase inferior vena caval pressure. This causes decreased cerebro spinal fluid (CSF) absorption which takes place in the lumbar cisterns. This increase in the CSF pressure is thereby transmitted causing increase in the ICP[2].

Bloomfield et al proved this hypothesis by abolishing the association between ICP and IAP by performing bilateral pleuropericardotomy with sternotomy in pigs. Cerebral perfusion pressure (CPP) is reduced as consequence of increased ICP.

CPP = MAP – IAP[99]

In patients with poltrauma with concommitent head and abdominal injuries, frequent monitoring of ICP, IAP and neurological symptoms and avoidance of hypervolemia are very crucial[2].

Certain recommendations laid down are :

1. In patients who are at risk of ICH or IAH, regular monitoring IAP (in all non-traumatic and traumatic patients)
2. Hypervolemia is avoided in patients with IAH to prevent increase in ICP.

3. In patients with IAH, frequent monitoring of the neurological statue is done.
4. Laparoscopy is avoided in patients with risk for ICH, as the pneumoperitonium created in experimental models had detrimental effects on the patient.

ABDOMINAL WALL

The blood flow to the abdominal wall is reduced by the direct compression effect of IAH, leading to localised ischemia and edema. This holds true for all the abdominal wall muscles. An increase in the IAP of only 10mmHg causes a 58% decrease of blood flow to the abdominis rectus sheath. This further worsens at 40mmHg[36]. Secondary to shock and fluid resuscitation, abdominal wall edema can occur which reduces the compliance of the abdominal wall muscles and further exacerbates IAH[2]. This may contribute to further complications such as wound dehiscence, impaired wound healing , necrotising fasciitis and herniation, especially in patients in whom the abdominal incision was closed under tension[36].

ABDOMINAL COMPARTMENT SYNDROME IN MORBIDLY OBESE PATIENTS

Baseline values of IAP have been found to be higher in obese individuals. In morbidly obese patients, elevated IAP has a far reaching effect on the organ function compared to their normal counterparts. Pseudotumour cerebri, hypo-ventilation syndrome, stress incontinence and gastro-esophageal reflux are now being attributed IAP in these obese patients. Further, increased incidence of incisional hernia and poor fascial healing rates have been related to increased IAP and the decrease of blood flow to the rectus sheath and the other muscles of the abdominal wall.

ABDOMINAL COMPARTMENTAL SYNDROME FOLLOWING LAPAROSCOPIC SURGERY

A rare complication of laparoscopic surgery is intestinal ischemia. Patients with atherosclerosis or cardiopulmonary disease are at a high risk. Proper pre-operative assessment to identify these patients is essential. Hence, a raised IAP due to pneumoperitoneum can predispose to splanchnic ischemia during laparoscopic surgery[50]. This causes decreased perfusion, mucosal acidosis and later acute intestinal distress syndrome. The increased IAP further compresses the splanchnic venous return reducing cardiac output directly. Moreover, the

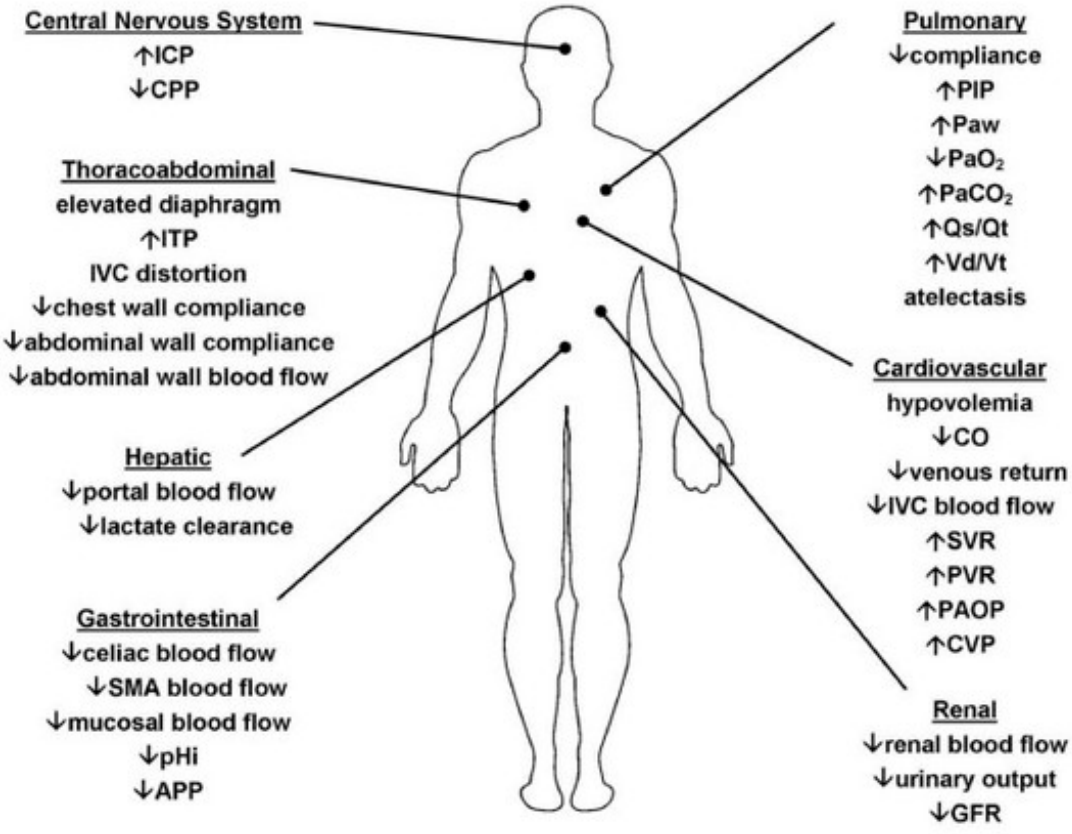
carbon dioxide (CO_2) is absorbed into the circulation, resulting in hypercapnia, respiratory acidosis, and increasing the systemic vascular resistance secondary to the hemodynamic stress response (anti-diuretic hormone, renin activity, and catecholamines) [50]. Acute intestinal distress syndrome may be further triggered by either significant vascular narrowing or arterial thrombosis as in our second case[50]

TREATMENT

NON-SURGICAL MANAGEMENT

It has been suggested by the WSACS, that the patients with two or more risk factors should have the IAP monitored at regular intervals. Treatment regimes are aimed to deal with two issues, organ support and lowering the intra abdominal pressure.

Fig 5: Summary of the vascular and pressure changes seen in IAH



MEDICAL MANAGEMENT OF IAH/ACS

1. Evacuation of intraluminal contents

   Ryles tube and suctioning

   Enemas

   Rectal tube

   Gastroprokinetics (cisapride, metaclopromide, erythromycin)

   Colonoprokinetics (prostigmine bolus or infusion, neostigmine)

   Endoscopic decompression of large bowel

   Ileostomy

   Colostomy

2. Evacuationof peri-intestinal and abdominal fluids

   CT or USG guided aspiration of abscess

   CT or USG guided aspiration of hematoma

   Percutaneous drainage of collection (blood)[2]

3. Improvement of abdominal wall compliance

   Sedation

   Pain relief

   Neuromuscular bloakage

   Body positioning

   Skin pressure decreasing interface

4. Correction of capillary leak and the positive fluid balance

   Correction of capillary leak

   Colloids instead of crystalloids

   Dobutamine

   Albumin in combination with diuretics (furosemide)

   Ascorbic acid in burns patient

   Dialysis or CVVH with ultrafiltration

5. Specific therapeutic interventions

   Continuous external abdominal pressure (CNAP)

   Negative external abdominal pressure (NEXAP)

   Targeted abdominal perfusion pressure (APP)

LOWERING INTRA-ABDOMINAL PRESSURE

Certain simple measures such as decompression of the stomach by passing nasogastric tube, supine positioning of the patient have modest effect in decreasing the intra abdominal pressure. Problems of aspiration are associated with the use of nasogastris tube. Hence the risk – benefit ratio should be considered.

Straining, coughing and ventilator dyssynchrony increase the intra abdominal pressure. Muscular paralysis for a period of time and sedation are beneficial. Reduced cardiac output as a complication of sedation, should be considered.

Enemas, prokinetic agents, flatus tube and aperients are used to decrease IAP. Some centers aim at reducing the IAP by draining fluid or gas by percutaneous or endoscopic decompression of the gastrointestinal tract.

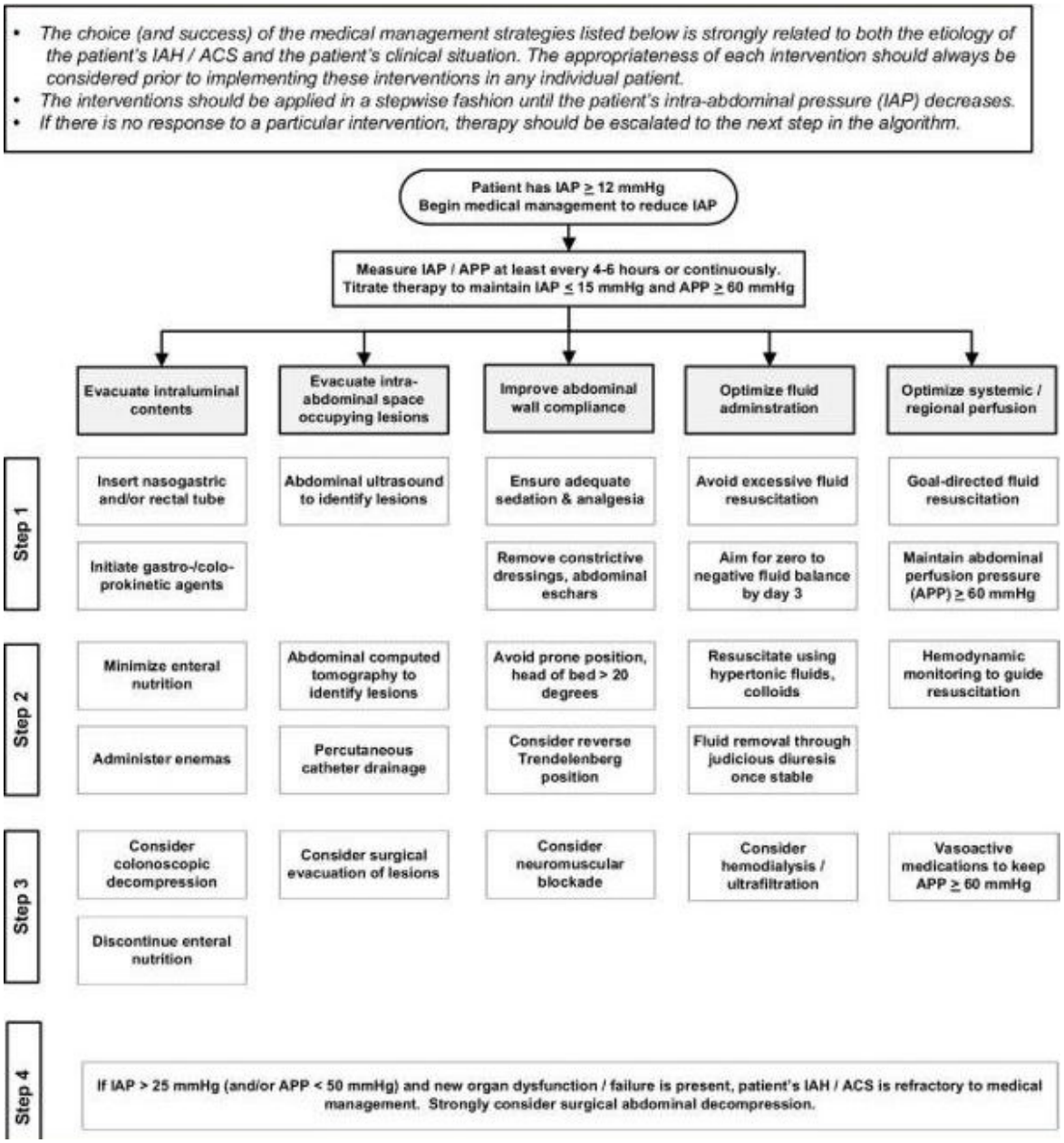


Fig 6: Flow chart – Medical management of IAH (Source : Am J Kidney Dis 2011 The National Kidney Foundation)

ORGAN SUPPORT

An important aim of management is to stabilize the cardiovascular system. As mentioned before, the problems affecting the cardiac status of the patient is more marked if the patient is hypovolemic. Hence initial resuscitation with optimal fluid to restore normovolemia is a simple but essential step in the management of the patient [37,38]. Prophylactic abdominal decompression has not gained popularity among general surgeons.

Optimizing clinical care would reduce adverse outcomes, which has been demonstrated in areas at the periphery of acute general surgery, such as pelvic fracture, where early hemorrhage control reduces the mortality significantly and in the process reduces the prevalence of ACS [2]. IAH exerts negative effects on colon healing and visceral blood flow[2]. Overloading the patient with fluid is detrimental. If the patient does not reach the 'target' APP of 60mmHg but is fluid replete, then inotropic and vasopressor agents are sought. There is no strict protocol as to which inotropic agent or vasopressor should be used first line. It should be tailored according the individual need of each patient.

Pulmonary artery wedge pressure and central venous pressure measurements are erroneous and should not be used to monitor the

patient's response. Due to increased ITP these values are falsely elevated. Monitoring values such as stroke value variation which is not altered by ITP is valuable in fluid resuscitation and management of the patient.

Biochemical and clinical signs of renal dysfunction should be considered in all patients. Renal replacement therapy may be started rather than fluid resuscitation with large volumes. There has been increasing incidences of secondary abdominal compartment syndrome due to ill management of fluid replacement. Gut integrity of the patient should be maintained by continuing entral feeding unless the patient is in primary or secondary gastrointestinal failure, causing unabsorption of the feed. Intra-abdominal pressure is not affected by the small volume of entral feed. A guide to the feed absorption is given by four hourly aspiration via the nasogastric tube. Adult respiratory syndrome should be prevented by using lung protective strategies.

The hepatic blood flow is altered, which leads to hepatic function deterioration and therefore drugs should be carefully considered before use. Moreover these patents are sensitive to the cardiac depressant action of the inducing anesthetic agent due to liver dysfunction and altered volume of distribution.

The combination of a sick, immobile patient with venous stasis increases the chances of venous thrombosis. Therefore prophylactic measures for venous thrombosis should be taken in these patients.

SURGICAL MANAGEMENT

In patients with abdominal compartment syndrome, improved mortality rates are seen following decompression of the abdomen by surgical intervention. If the abdomen is left open, it would lead to the exposure of the bowel to air causing their drying and dessication. Hence to increase the intra abdominal space a plastic/ Bagota bag is used and stitched to the wound edges. This recreates a closed abdominal cavity with increased volume thereby decreasing the pressure in the abdominal cavity. Continuous/ regular monitoring of the intra-abdominal pressure should be done in these patients.

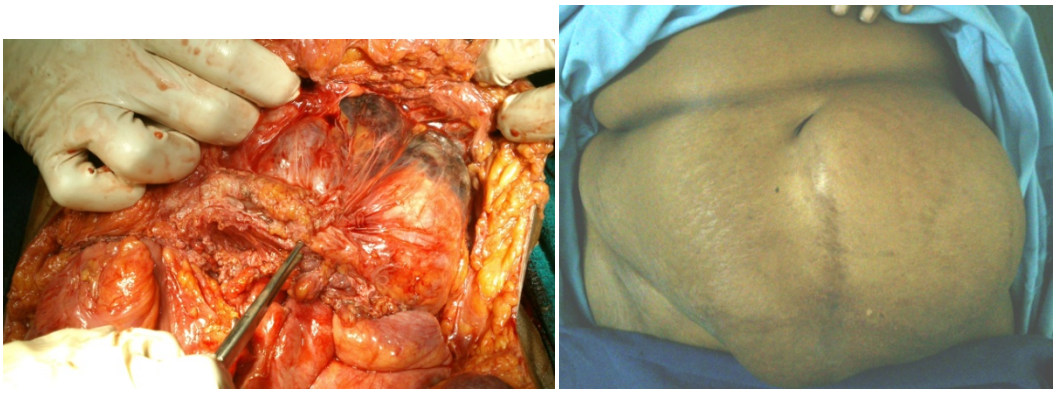


Fig 7, 8: Massive incisional hernia

Another method which has been used to treat patients with increased IAP is subcutaneous release of the linea alba, leaving the peritoneum and the skin intact. The same principle applies here where the intra abdominal volume has been increased causing the decrease in the intra abdominal pressure. This is mainly done for pancreatitis patients in the acute setting.

The Bagota bag is mainly used in two settings,

(1) where the medical management of decreasing intra abdominal pressure has failed and the patient needs emergency reduction in the IAP for preserving the bowel viability.

(2) In patients who have a high risk of developing ACS, the primary modality of treatment is temporary closure with Bagota bag[45].

The management options for open abdomen include cutaneous advancement flap (Skin only) closure, split thickness skin grafting, vacuum assisted closure techniques (without or with retention suture), zipper system, Bagota bag, synthetic mesh, sandwich method, silicone rubber sheet and occlusive dressing under suction[40-43]. Complications are associated with each of these techniques, retraction of abdominal fascia, bowel fistula formation and intestinal adherence to the prosthesis[2]. Enteroatmospheric fistula and skin excoriation are common complications of Bagota bag. Also the quantification of peritoneal fluid

cannot be done due to its leak around the wound edges. Sterile human chorioamniotic membrane has been used as an alternative to Bagota/ plastic bags[43,45]. Complications of fistula and serosal erosions have come down.

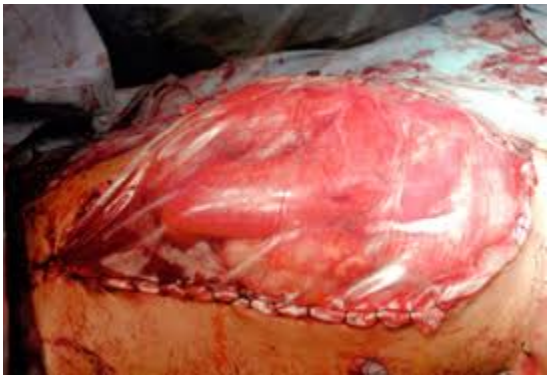


Fig 9: Bagota Bag application following emergency laparotomy for massive incisional hernia

In cases of zipper fastners, requires the prosthetic material to be sutured to the abdominal wall tissue. The advantage being it has less adherence to the viscera underlying it. The problems underlying its use are,

1. Management of the peritoneal fluid unless a drain is provided
2. Difficulty in reapplication once zipper is applied.
3. Leak of peritoneal fluid which soak the dressings on closure of the skin

Fig10 : Figure illustrating zipper technique

Sandwich technique is generally well tolerated in critically ill patients and easy to construct. Disadvantages are,

1. Recurrence of ACS, due to increase in IAP following application of sutures in the fascial edges
2. Fascial necrosis in abdomen which had been left open

Polypropylene mesh placement as a means of temporary abdominal closure. It can be used either with or without zipper[2]. Zipper is used in cases of expected abdominal re-exploration. Disadvantages

1. Adherence of the underlying viscera to the mesh and subsequent injury to the organ following re-exploration
2. Mesh may erode into the bowel if left long enough
3. Fascial necrosis may occur following repetitive suturing of the biosynthetic material to the fascial edges.

Occlusive dressing under suction is an easy procedure with less technical problems. In case of peritoneal fluid leak, bedside additional adhesive drapes are applied over the leak site. This has been replaced by Vacuum assisted closure (VAC) techniques

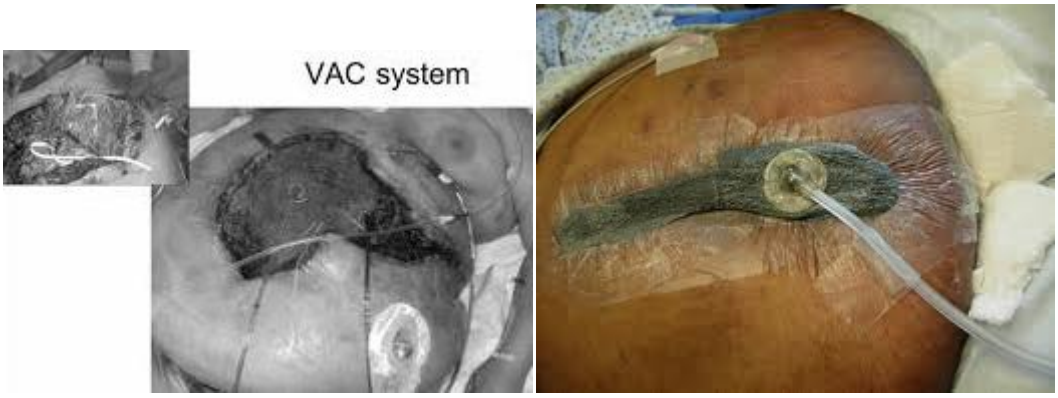


Fig 11: Figure illustrating Vacuum Assisted Closure technique

Advantages of VAC technique are,

1. Avoids mechanical damage to the abdominal wall tissues as it consists of sutureless closure.
2. Quantification and replacement of the peritoneal fluid can be done.
3. Risk of bowel injury is reduced, especially in times of re-exploration.
4. Adhesive-backed drapes stabilise and seal the wound edges. The passage of fluid in and out of the wound is prevented.

5. Skin soilage is minimised and surrounding skin is protected
6. Tissue necrosis and infection were not observed in VAC patients[44]

On average, the abdomen is closed five days after the decompression, by when the risk of intra abdominal hypertension has usually passed. When surgical closure is not possible, vacuum dressing aiding in wound closure has shown good results.

In critically ill patients with ACS there are four key concerns

PHARMACODYNAMICS/KINETICS

The cardiac depressant effects of inducing agents are more exaggerated due to altered drug handling, liver dysfunction, hypovolaemia and altered volume of distribution in patients with ACS. Hence careful inducton and reduced dose of drugs to be given with intensive monitoring of patient is required.

SUDDEN DECREASE IN INTRA-THORACIC PRESSURE

On opening the abdomen during laparotomy, the intra-abdominal pressure rapidly equilibrates with the atmospheric pressure. This causes the ITP to consequently decrease causing the respiratory compliance to

increase dramatically. Lung parenchymal damage due to barotrauma and volutrauma with potential 'over ventilation' may occur. Therefore close attention to tidal volume and airway pressure should be paid.

SUDDEN DECREASE IN SYSTEMIC VASCULAR RESISTANCE

The IAP comes to atmospheric pressure on opening the abdomen. This causes the afterload to fall dramatically and so may the arterial pressure due to fall in the cardiac output. Hence resuscitative drugs and equipment should be available at any point of time. Fluid loading with or without vasopressors may also be required.

REPERFUSION INJURY

Ischemia-reperfusion injuries may occur following laparotomy as previously ischemic viscera and parts of bowel get reperfused causing complications such as arrhythmias, myocardial depression and cardiac arrest.

Extreme care and vigilance at the time of anesthetic induction and opening the abdomen should be taken. Resuscitaion equipments and drugs should be available at all times.

THE MANAGEMENT PROTOCOL FOR INTRA-ABDOMINAL HYPERTENSION/ ABDOMINL COMPARTMENT SYNDROME AS RECOMMENDED BY WORLD SOCIETY OF THE ABDOMINAL COMPARTMENT SYNDROME

1. Patients on ICU admission should be screened for independent risk of IAH/ACS
2. In the presence of two or more risk factors, baseline IAP is measured
3. If IAH is present, serial IAP monitoring is done which helps in guiding patient's resuscitation
4. The main aim is that APP should be maintained above 60mmHg
5. Ventillator dys-synchromy, pain and agitation – cause abdominal muscles to contract, increasing their tone and IAP. Also accessory muscles of respiration are used which increases the tone.
6. A trial of sedation and neuromuscular bloakade in cases of mild IAH, helps in muscle relaxation and decreasing IAP. Neuromuscular blockade useful in cases of third-space fluid and tight abdominal closure.
7. Positioning of the patient – supine. Head end of bed elevation causes increase in IAP

8. Nasogastric tube/ enemas/rectal tubes/ prokinetic agents/ endoscopic decompression are useful as both air and fluid within the hollow visera increase IAP
9. Fluid resuscitation should be optimal as overhydration leads to secomdary abdominal compartment syndrome and hypovolaemia causes exaggeration of complications, esp cardiac, hepatic and renal
10. Percutaneous catheter decompression in cases of intraperitoneal abscess, blood or fluid in symptomatic ACS under ultrasound guidance.
11. Open abdominal decompression in selected patients not responding to medical management and those unfit for percutaneous drainage.

In “open abdomen” management – cutaneous advancement flap / split thickness skin graft/ vacuum assisted closure techniques zipper, Bagota bag, synthetic mesh, silicon rubber sheets and occlusive dressing under suction and sterilised human chorioamniotic membrane have been tried.

Open abdomen in a complex clinical problem. Newer techniques and technologies have been developed which now allow improved management and progressive reduction of the fascial defect[123]. Recent

studies have shown that most of the patients treated as open abdomen are closed within their initial hospitalisation. Several techniques for fascial closure are available now[2]. Squential fascial closure[47], split thickness skin grafting to cover the exposed bowel[124], biological or prosthetic mesh approximation of the bowel, component separation, abdominal reapproximation anchor (ABRA) [48]and anterior rectus abdominis sheath turnover flap.

Management of open abdomen and its core principle should be essentially understood by trauma and general surgeons. Open Abdomen Advisory Panel was established for this purpose[49]. Certain principles and recommendations were established by them on comprehensive evidence based management strategy. It recommends that in high risk patients, early use of an open abdomen improves the survival from ACS. The improvement is associated with increased numbers of primary closure of the abdomen and not on increased resource utilisation[2]. The optimal management techniques are still to be established by the ongoing prospective trials.

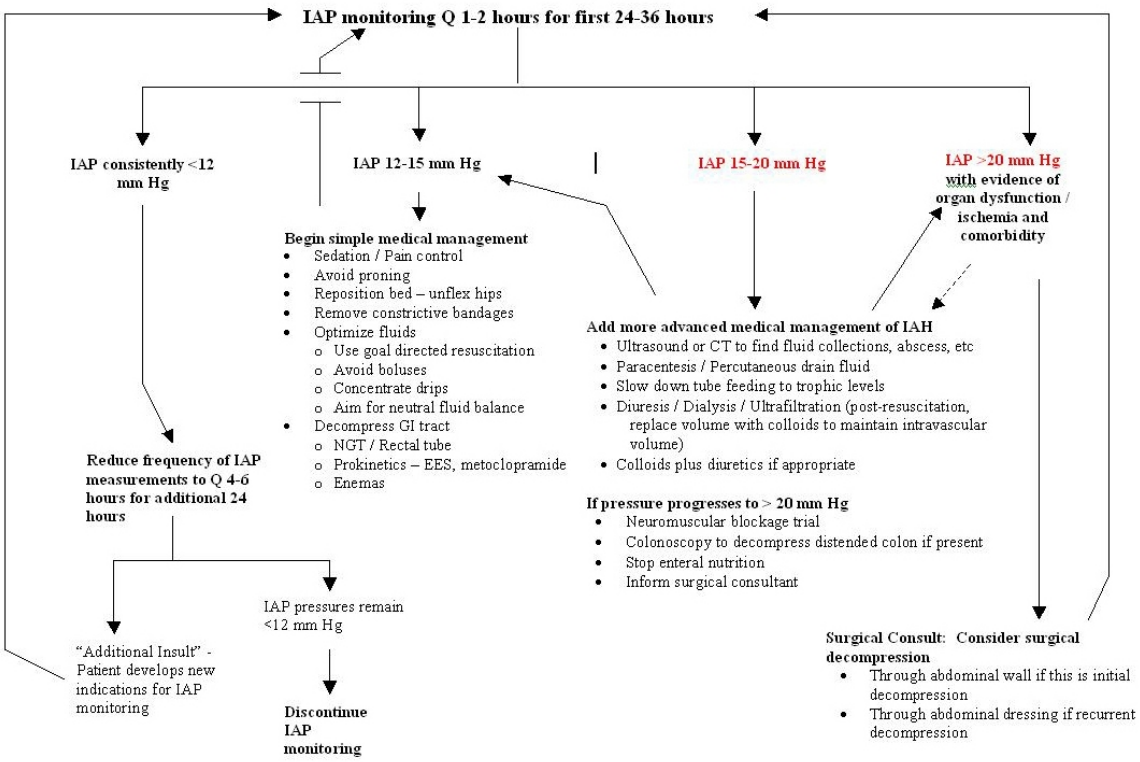


Fig 12: Schematic diagram illustrating Intra-abdominal pressure monitoring algorithm

AIM OF THE STUDY

To assess the relationship between peri operative intra abdominal pressure and post operative patient morbidity, (with special reference to kidney function and pulmonary atelectasis) , and mortality.

MATERIAL AND METHODS

The study was be conducted on 50 cases of Emergency Laparotomy and 50 elective laparotomy patients admitted in Government Royapettah Hospital attached to KILPAUK MEDICAL COLLEGE AND HOSPITAL. Ethical committee clearance was obtained from the institutional ethical committee of KILPAUK MEDICAL COLLEGE AND HOSPITAL. This study was conducted between May2013 and December 2013. Before the study conducted informed consent was obtained from all the patients.

METHOD OF COLLECTION

Study design : A Prospective, Comparative study.

Sample size : 100 admitted cases

Inclusion criteria :

1. patients admitted to GRH and undergoing emergency laparotomy
2. Age group 18 yrs to 60 yrs

Exclusion criteria :

1. Age group < 18yrs and > 60yrs
2. Pregnancy
3. Morbid obesity
4. Spinal cord problems and fracture limbs who are unable to lie down supine
5. Bladder complains – neurogenic bladder, cystitis
6. Patients with established causes of co-morbidity such as renal failure, CAD, hypertension

METHODOLOGY:

Two groups of 50 surgical patients were selected. The experimental group had increased intra-abdominal pressure. The control group were selected from those undergoing elective surgery. In both groups, patients with factors which could cause increased intra-abdominal pressure like obesity were excluded.

The preliminary details collected from patients included, name, age, sex, diagnosis, operative procedure planned, BMI. Presence or absence of diabetes mellitus, hypertension. Examination findings of pulse, blood pressure, respiratory rate, temperature and specific systemic examination of respiratory system, cardiovascular system and abdomen were noted.

Laboratory tests done preoperatively and post operatively according to need were hemoglobin concentration, plasma urea, plasma creatinine and Chest X-Ray. Intra-abdominal pressure and urine output were monitored pre-operatively and at four hourly intervals in the post-operative period for 24 hours.

The intravesical route of measuring the IAP was done by catheterization of the urinary bladder using Foley's attached to a three way tap which was then connected to a pressure transducer. The patient was placed in a supine position and the residual urine was drained. The catheter was connected to a pressure transducer and the point of mid-axillary line at the iliac crest was taken as the reference point where the intra-abdominal pressure value was zeroed. Around

25ml (if weight <20kgs, 1ml per kg) of saline was instilled into the bladder. The reading was taken one minute later, providing time for detrusor muscle relaxation. The measurements were taken in the absence of active abdominal muscle contraction and at the end of expiration. Measurements were made at regular intervals, usually varying from 4 to 6hrs. Depending on the IAP the treatment modality was adjusted. The Foleys catheter was clamped before each measurement.

Operative findings during laparotomy and the surgical procedure done were noted. Post-operative mechanical ventilation and re-laparotomy were followed up if needed. Also, cause of mortality was noted if the patient expired in spite of maximal supportive medical care.

The results were documented and tabulated. The statistical significance was found out using the Chi square test.

Fig13 : :Equipment used

Fig14: Intra abdominal pressure monitoring – GRH

RESULTS AND OBSERVATIONS

This study was conducted on 50 cases of Emergency Laparotomy and 50 elective laparotomy patients admitted to Government Royapettah Hospital (GRH) attached to KILPAUK MEDICAL COLLEGE AND HOSPITAL. Data was collected between May2013 and December 2013. The mean (S.D.) intravesical pressure of patients who underwent emergency laparotomy and those having elective surgery was 16.6 (4.16) and 4.22 (2.46) respectively. These groups are called Increased IAP group and Normal IAP group hereafter.

Table 1: Sex * Group Crosstabulation

			Group		Total	P value
			Normal IAP	Increased IAP		
Sex	Male	Count	21	41	62	<0.001
		% within Sex	33.9%	66.1%	100.0%	
		% within Group	42.0%	82.0%	62.0%	
	Female	Count	29	9	38	
		% within Sex	76.3%	23.7%	100.0%	
		% within Group	58.0%	18.0%	38.0%	
Total		Count	50	50	100	
		% within Sex	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

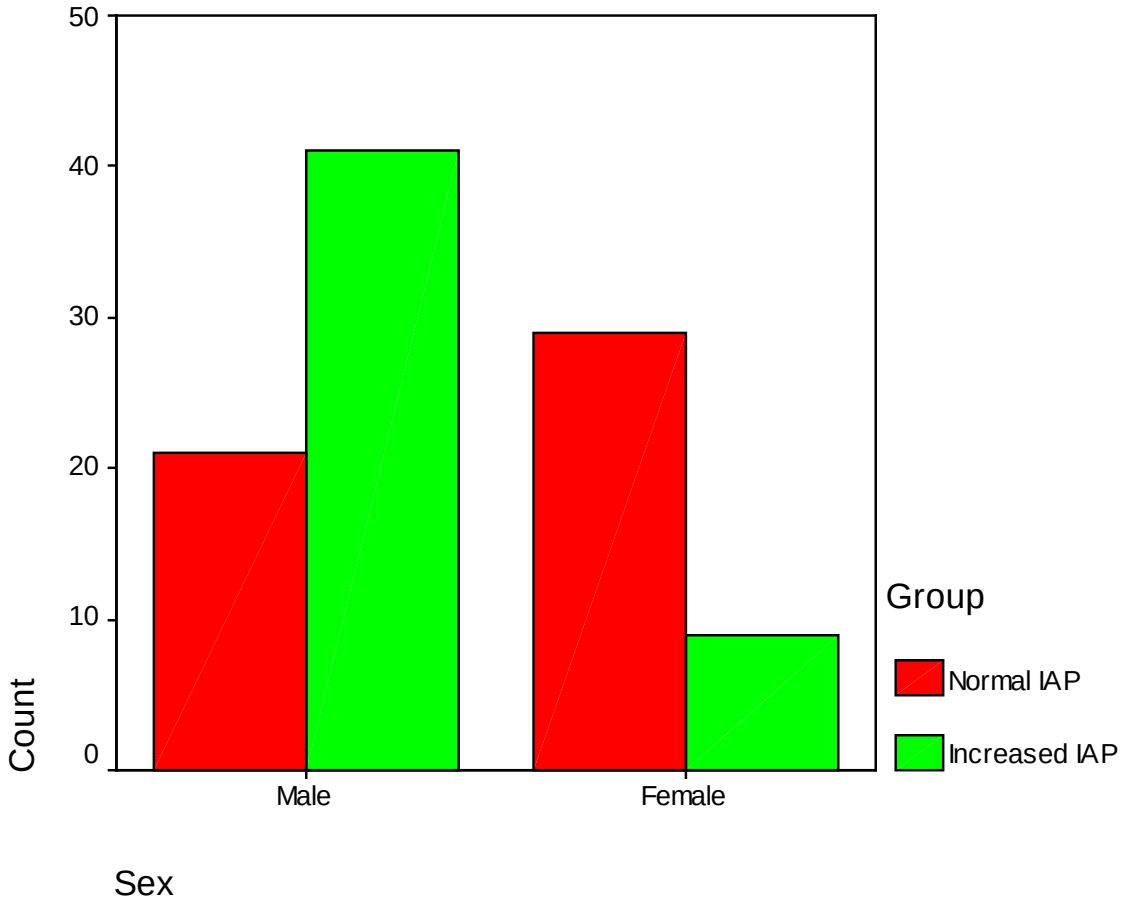


Figure 15: Gender distribution

The incidence of increased intra-abdominal pressure (emergency) is significantly more among men than in women ($p<0.001$). The number of female patients is more in the elective surgery list. Hence more number of males suffered from intra abdominal hypertension in the study group.

Table 2: Age *Group Crosstabulation

	Group	N	Mean	Std. Deviation	P value
Age in years	Normal IAP	50	46.20	13.737	0.149
	Increased IAP	50	41.98	15.236	

The mean age between the two groups is given above and does not show any significant difference.

Table 3: BMI * Group Crosstabulation

			Group		Total	P value
			Normal IAP	Increased IAP		
BMI	Underweight	Count	1	0	1	0.396
		% within BMI	100.0%	.0%	100.0%	
		% within Group	2.0%	.0%	1.0%	
	Normal	Count	30	35	65	
		% within BMI	46.2%	53.8%	100.0%	
		% within Group	60.0%	70.0%	65.0%	
	Overweight	Count	19	15	34	
		% within BMI	55.9%	44.1%	100.0%	
		% within Group	38.0%	30.0%	34.0%	
Total		Count	50	50	100	
		% within BMI	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

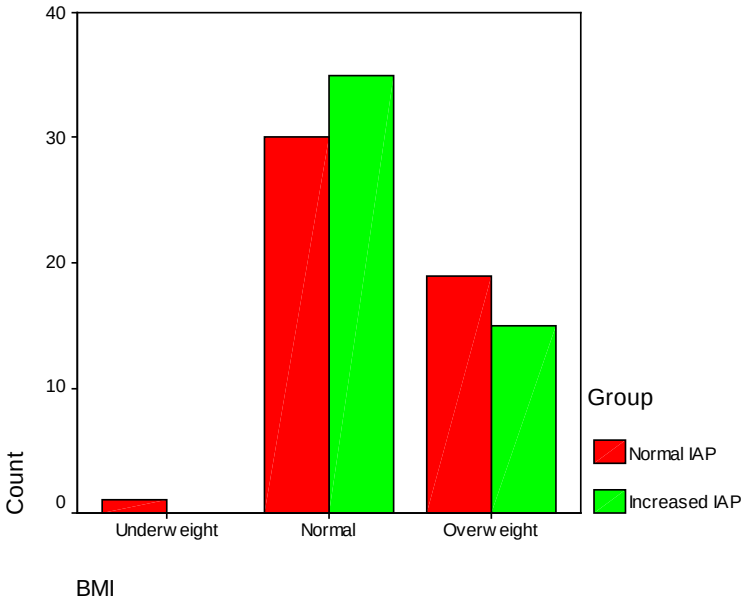


Figure 16: BMI distribution chart

There is no significant difference in the prevalence of cases based on the BMI. This suggests that's the BMI and hence over weight of the patient does not significantly increase the intra abdominal pressure of the patient to critical level so as to cause intra abdominal hypertension.

Table 4: Group Statistics-Urea levels in patients with normal and increased abdominal pressure

	Group	N	Mean	Std. Deviation	Std. Error Mean	P value
Urea – pre	Normal IAP	50	33.54	5.970	.844	<0.001
	Increased IAP	50	47.82	11.261	1.593	
Urea – op	Normal IAP	50	32.44	4.807	.680	<0.001
	Increased IAP	50	44.78	10.533	1.490	
Urea - 4h	Normal IAP	50	32.96	5.103	.722	<0.001
	Increased IAP	50	44.04	10.103	1.429	
Urea - 12h	Normal IAP	50	33.28	4.481	.634	<0.001
	Increased IAP	50	42.90	9.179	1.298	
Urea - 24h	Normal IAP	50	32.74	3.848	.544	<0.001
	Increased IAP	50	42.50	9.554	1.351	

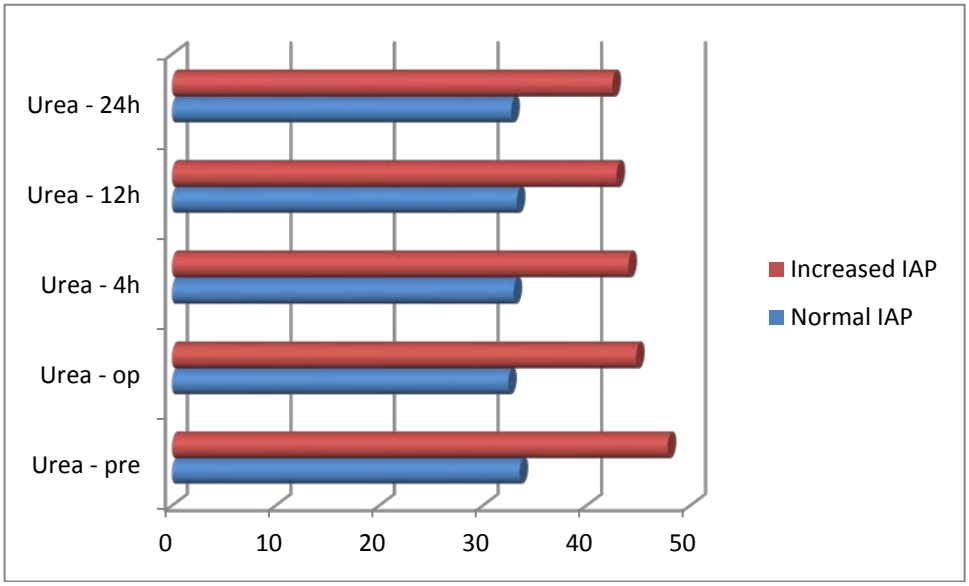


Fig 17: Urea levels in patients with normal and increased abdominal pressure - Group Statistics

Urea is significantly raised in all the cases of increased intra-abdominal pressure as compared to the cases with normal intra-abdominal pressure in all time intervals (pre-operative, post – operative, 4th hourly, 12hourly, 24hourly, indicating renal dysfunction in cases of increased intra abdominal pressure.

Table 5: Group Statistics- Creatinine levels in patients with normal and increased IAP

	Group	N	Mean	Std. Deviation	Std. Error Mean	P value
Cre – pre	Normal IAP	50	1.052	.2667	.0377	<0.001
	Increased IAP	50	1.838	1.2660	.1790	
Cre – op	Normal IAP	50	.980	.2740	.0388	<0.001
	Increased IAP	50	1.594	1.1474	.1623	
Cre - 4h	Normal IAP	50	1.064	.2776	.0393	0.001
	Increased IAP	50	1.588	1.0578	.1496	
Cre - 12h	Normal IAP	50	1.042	.2425	.0343	0.001
	Increased IAP	50	1.548	.9725	.1375	
Cre - 24h	Normal IAP	50	.996	.2432	.0344	0.001
	Increased IAP	50	1.460	.8947	.1265	

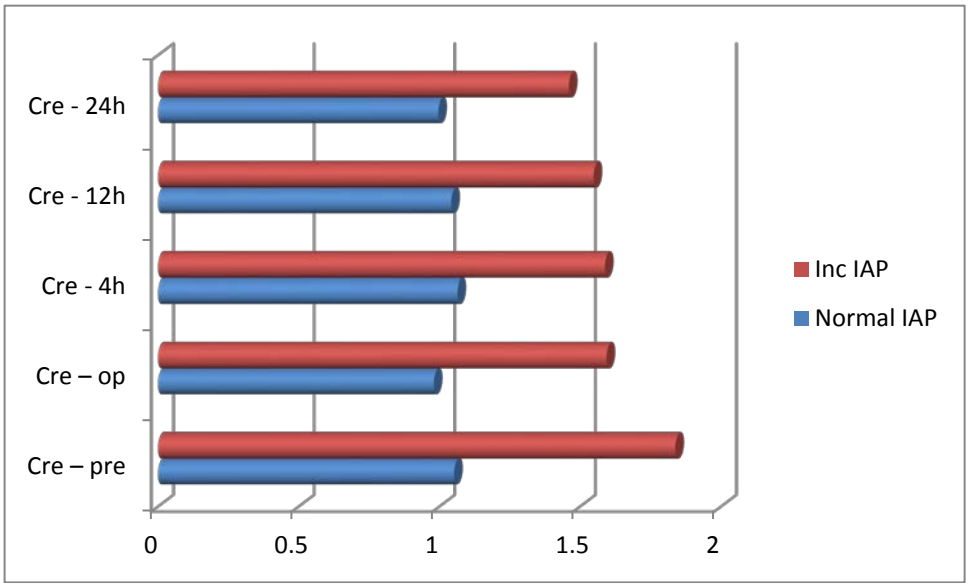


Figure 18: Creatinine levels in patients with normal and increased IAP-

Group Statistics

There is found to be a significant increase in the creatinine level in all cases of increased intra-abdominal pressure as compared to the normal group at all time intervals. This similarly indicates renal dysfunction in cases of increased intra abdominal pressure.

Table 6: Group Statistics – Urine output in patients with normal and increased IAP

	Group	N	Mean	Std. Deviation	Std. Error Mean	P value
Urine - 4h	Normal IAP	50	303.50	123.616	17.482	0.175
	Increased IAP	50	270.00	121.323	17.158	
Urine - 8h	Normal IAP	50	258.00	93.055	13.160	0.843
	Increased IAP	50	263.00	152.382	21.550	
Urine - 12h	Normal IAP	50	270.50	94.988	13.433	0.243
	Increased IAP	50	247.00	104.569	14.788	
Urine - 16h	Normal IAP	50	243.50	95.406	13.492	0.256
	Increased IAP	50	274.00	162.644	23.001	
Urine - 20h	Normal IAP	50	263.50	96.258	13.613	0.782
	Increased IAP	50	269.50	118.697	16.786	
Urine - 24h	Normal IAP	50	254.00	82.894	11.723	0.565
	Increased IAP	50	266.00	121.193	17.139	

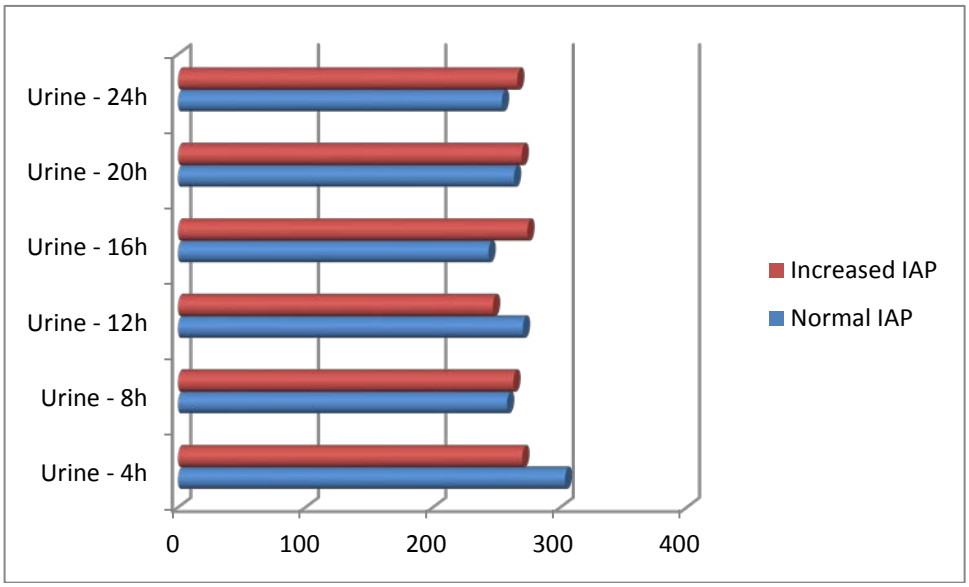


Figure 19: Urine output in patients with normal and increased IAP- Group Statistics

There is no significant correlation between the urine output and the intra-abdominal pressure. This might indicate that urine output might not be an ideal indicator of renal dysfunction in cases of intra abdominal hypertension or abdominal compartment syndrome.

Table 7: Pre-operative * Group Atelectasis - Crosstabulation

			Group		Total	P value
			Normal IAP	Increased IAP		
Atelectasis - Pre	Yes	Count	0	3	3	0.079
		% within Atelectasis - Pre	.0%	100.0%	100.0%	
		% within Group	.0%	6.0%	3.0%	
	No	Count	50	47	97	
		% within Atelectasis - Pre	51.5%	48.5%	100.0%	
		% within Group	100.0%	94.0%	97.0%	
Total		Count	50	50	100	
		% within Atelectasis - Pre	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

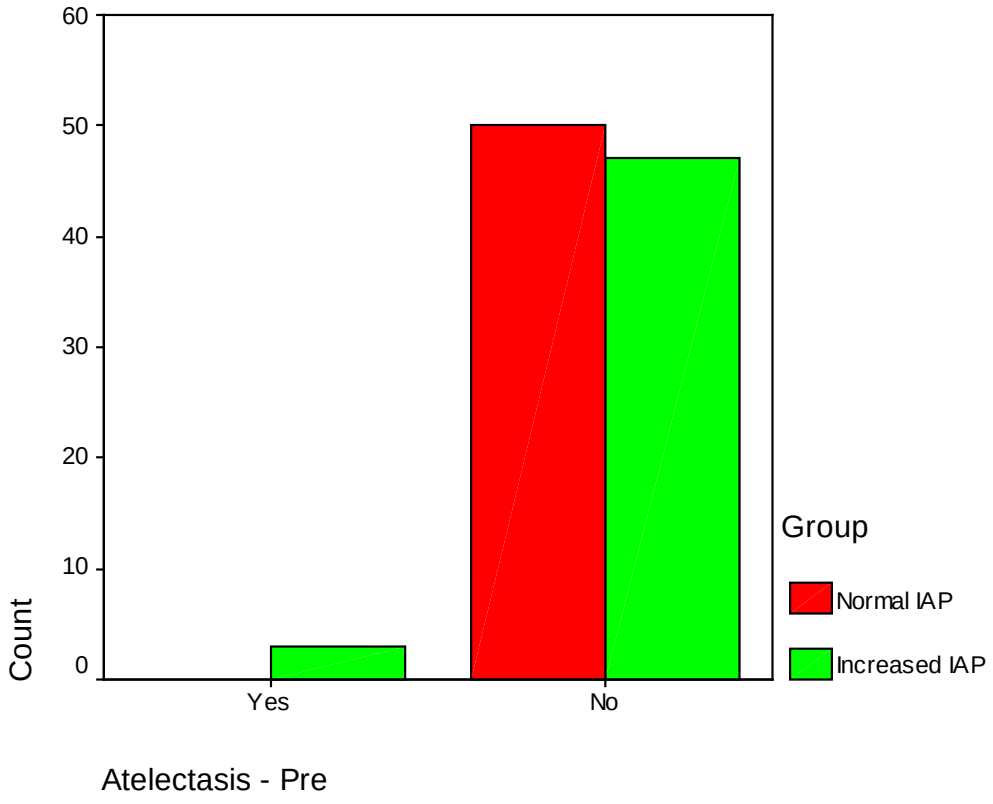


Figure 20: Pre-operative * Group Atelectasis

There is no significant difference in the incidence of atelectasis pre-operatively between the two groups.

Table 8 : Post-operative * Group Atelectasis - Crosstabulation

			Group		Total	P value
			Normal IAP	Increased IAP		
Atelectasis – Post	Yes	Count	4	12	16	0.027
		% within Atelectasis – Post	25.0%	75.0%	100.0%	
		% within Group	8.0%	24.0%	16.0%	
	No	Count	46	38	84	
		% within Atelectasis – Post	54.8%	45.2%	100.0%	
		% within Group	92.0%	76.0%	84.0%	
Total		Count	50	50	100	
		% within Atelectasis – Post	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

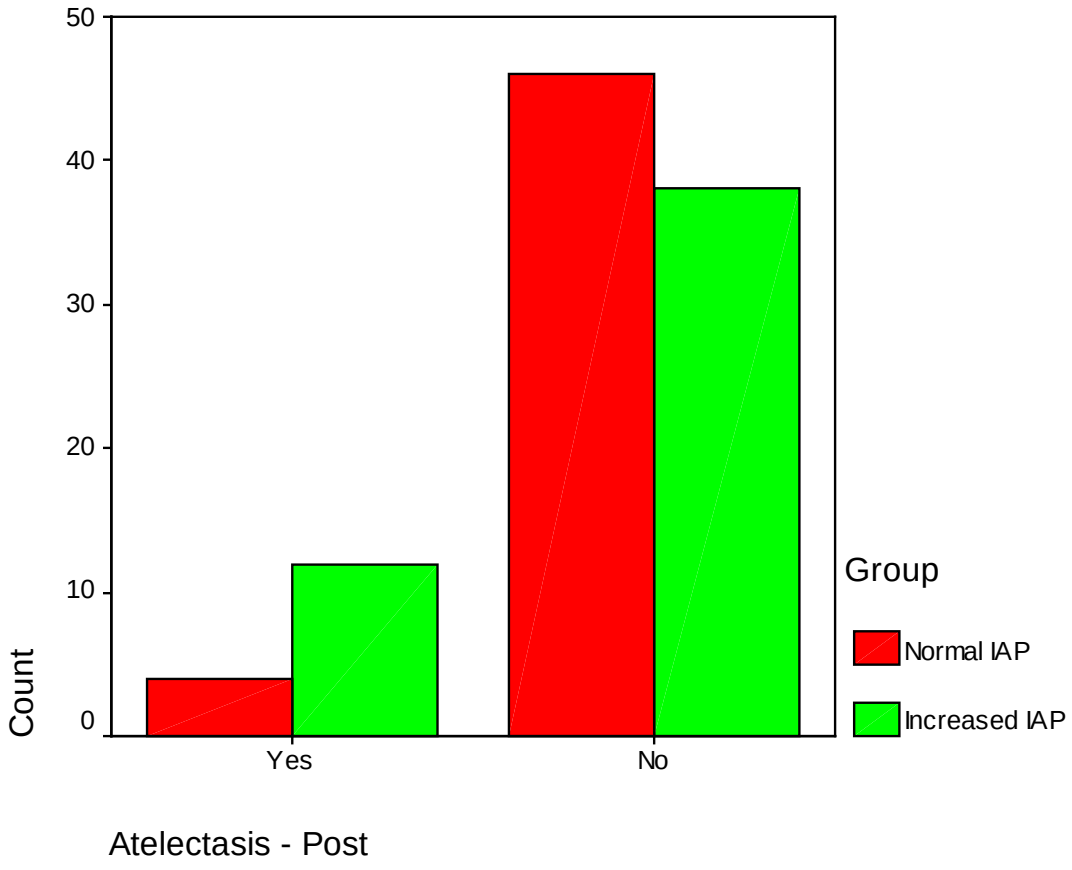


Figure 21: Post-operative * Group Atelectasis

There is a mild increase in the incidence of atelectasis post-operatively in cases of increased intra-abdominal pressure as compared to the group with normal intra abdominal pressure. This might indicate that pulmonary atelectasis correlation is to the duration of intra abdominal hypertension as the post operative intra abdominal pressure is more in the emergency cases than the elective cases.

Table 9: Post-op ventillation * Group - crosstabulation

			Group		Total	P value
			Normal IAP	Increased IAP		
Post-op ventillation	Yes	Count	3	4	7	0.695
		% within Post-op ventillation	42.9%	57.1%	100.0%	
		% within Group	6.0%	8.0%	7.0%	
	No	Count	47	46	93	
		% within Post-op ventillation	50.5%	49.5%	100.0%	
		% within Group	94.0%	92.0%	93.0%	
Total		Count	50	50	100	
		% within Post-op ventillation	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

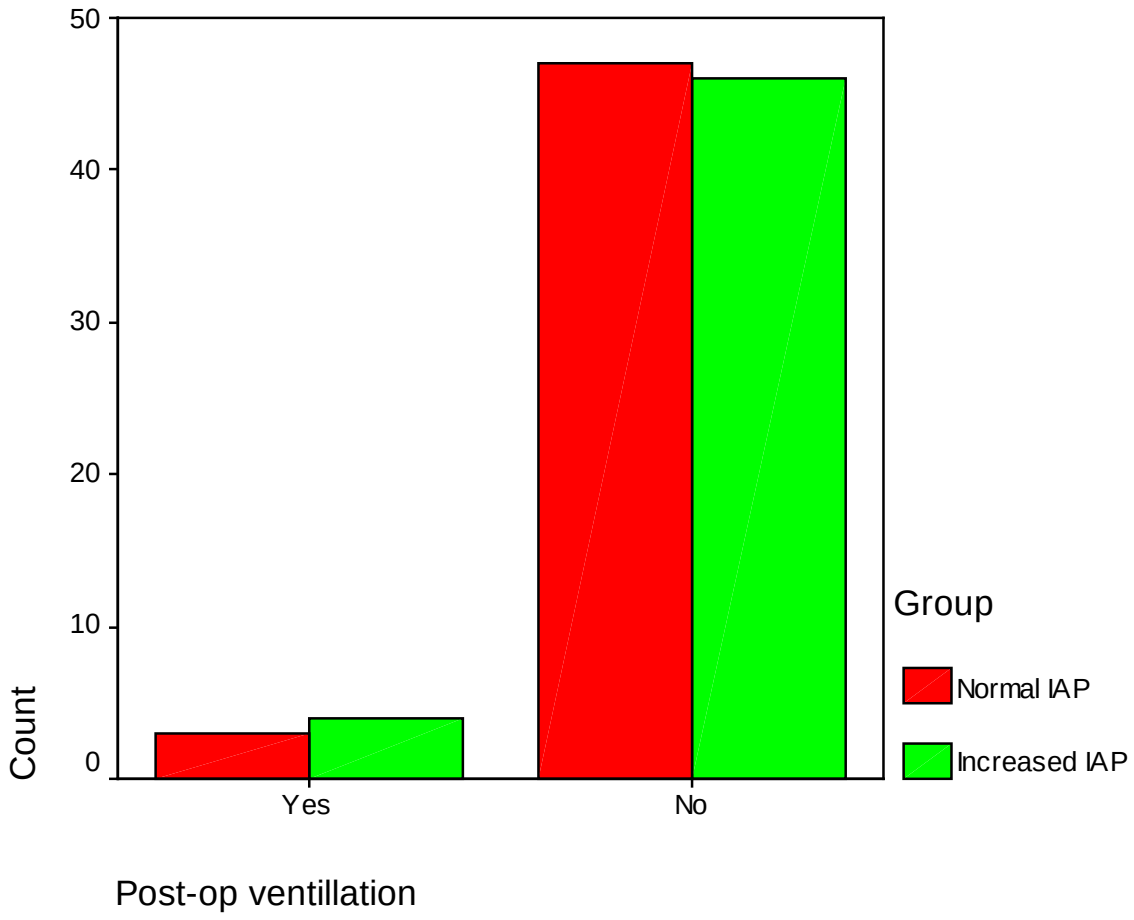


Figure 22 : Post-op ventillation * Group distribution

There is no significant difference in the post operative need for mechanical ventilation among the two groups

Table 10: Re-laparotomy * Group Crosstabulation

			Group		Total	P value
			Normal IAP	Increased IAP		
Re-laparotomy	Yes	Count	0	1	1	0.315
		% within Re-laparotomy	.0%	100.0%	100.0%	
		% within Group	.0%	2.0%	1.0%	
	No	Count	50	49	99	
		% within Re-laparotomy	50.5%	49.5%	100.0%	
		% within Group	100.0%	98.0%	99.0%	
Total		Count	50	50	100	
		% within Re-laparotomy	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

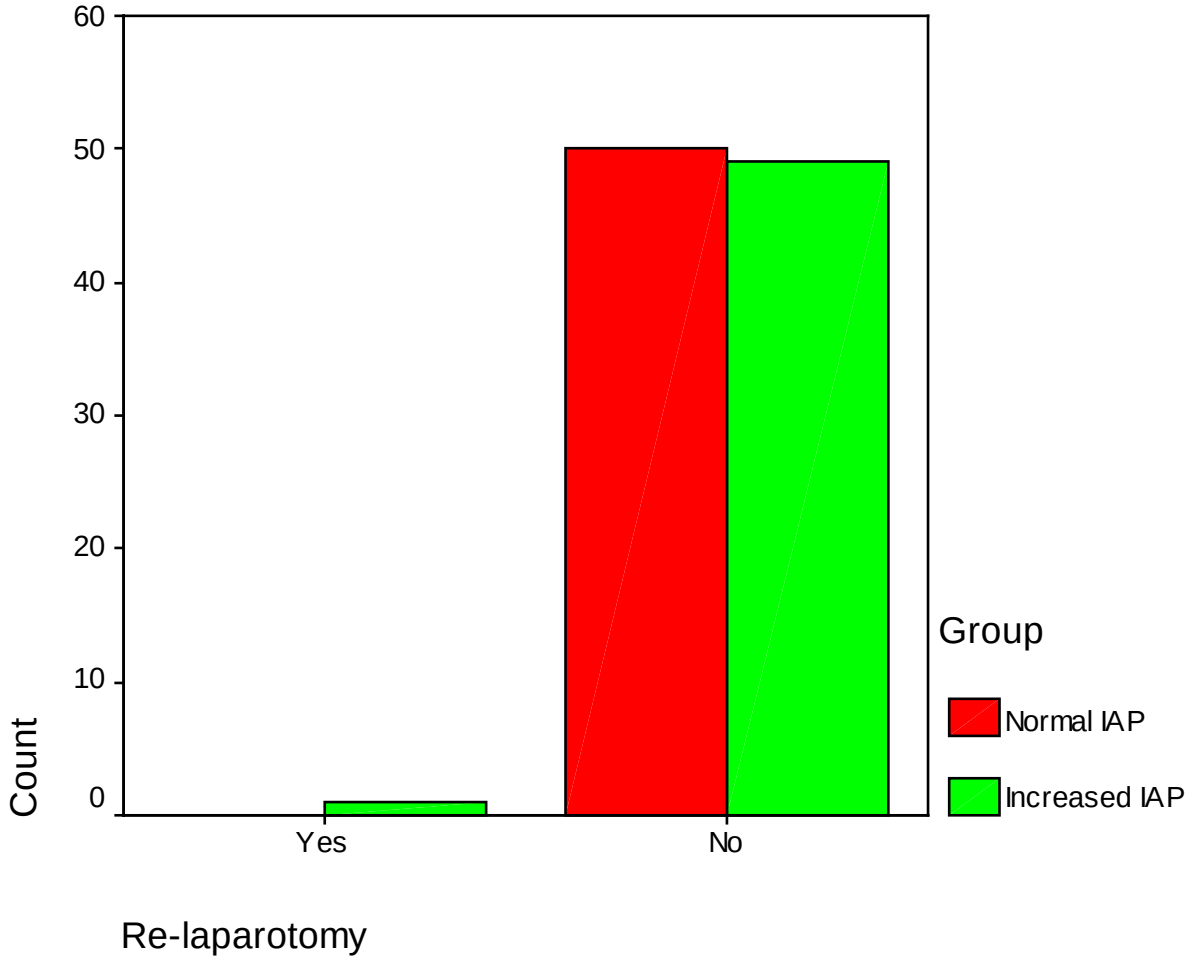


Figure 23: Re-laparotomy * Group distribution

There is no significant difference in the need for re-laparotomy among the two groups

Table 11: IAP and Urea levels Emergency laparotomy*Group - Crosstabulation

		Urea - pre	Urea – op	Urea - 4h	Urea - 12h	Urea - 24h
Iap – pre	Pearson Correlation	.152	.104	.066	.043	.006
	Sig. (2-tailed)	.294	.473	.651	.768	.969
	N	50	50	50	50	50
Iap – op	Pearson Correlation	.239	.168	.125	.139	.055
	Sig. (2-tailed)	.094	.243	.389	.334	.705
	N	50	50	50	50	50
Iap - 4h	Pearson Correlation	.224	.141	.075	.099	.019
	Sig. (2-tailed)	.119	.328	.607	.494	.895
	N	50	50	50	50	50
Iap - 8h	Pearson Correlation	.214	.149	.067	.105	.022
	Sig. (2-tailed)	.136	.301	.641	.467	.878
	N	50	50	50	50	50
Iap - 12h	Pearson Correlation	.183	.122	.047	.092	.008
	Sig. (2-tailed)	.203	.398	.746	.527	.954
	N	50	50	50	50	50
Iap - 16h	Pearson Correlation	.178	.104	.039	.082	.000
	Sig. (2-tailed)	.216	.471	.790	.572	.999
	N	50	50	50	50	50
Iap - 20h	Pearson Correlation	.163	.079	.015	.057	-.045
	Sig. (2-tailed)	.259	.585	.916	.696	.759
	N	50	50	50	50	50
Iap - 24h	Pearson Correlation	.147	.072	.006	.048	-.046
	Sig. (2-tailed)	.307	.622	.966	.743	.752
	N	50	50	50	50	50

This is the cross tabulation between the intra abdominal pressure pre operatively and post operatively at fourth hourly interval for the first 24 hours and the respective urea levels at these intervals. There seems to be no correlation between them even though earlier it was evident that urea level was relatively higher in patients with increased intra abdominal pressure than ones with normal. Hence it reinforces the concept that, the duration of increased intra abdominal pressure is more significant than the actual pressure.

Table 12: IAP and creatinine levels Emergency laparotomy *Group – Crosstabulation

		Cre - pre	Cre - op	Cre - 4h	Cre - 12h	Cre - 24h
Iap – pre	Pearson Correlation	.104	.118	.149	.131	.120
	Sig. (2-tailed)	.471	.416	.301	.364	.408
	N	50	50	50	50	50
Iap – op	Pearson Correlation	.127	.180	.223	.201	.181
	Sig. (2-tailed)	.379	.212	.120	.161	.209
	N	50	50	50	50	50
Iap - 4h	Pearson Correlation	.135	.169	.220	.196	.175
	Sig. (2-tailed)	.350	.241	.125	.172	.225
	N	50	50	50	50	50
Iap - 8h	Pearson Correlation	.128	.151	.207	.185	.164
	Sig. (2-tailed)	.376	.295	.150	.199	.256
	N	50	50	50	50	50
Iap - 12h	Pearson Correlation	.114	.136	.196	.176	.159
	Sig. (2-tailed)	.431	.347	.174	.222	.271
	N	50	50	50	50	50
Iap - 16h	Pearson Correlation	.101	.134	.198	.176	.155
	Sig. (2-tailed)	.484	.354	.169	.220	.281
	N	50	50	50	50	50
Iap - 20h	Pearson Correlation	.123	.154	.224	.212	.194
	Sig. (2-tailed)	.393	.286	.118	.140	.178
	N	50	50	50	50	50
Iap - 24h	Pearson Correlation	.107	.139	.199	.190	.162
	Sig. (2-tailed)	.460	.337	.165	.187	.261
	N	50	50	50	50	50

This is the cross tabulation between the intra abdominal pressure pre operatively and post operatively at fourth hourly interval for the first 24 hours and the

respective creatinine levels at these intervals. There seems to be no correlation between them even though earlier it was evident that creatiine level was relatively higher in patients with increased intra abdominal pressure than ones with normal. Hence it reinforces the concept that, the duration of increased intra abdominal pressure is more significant than the actual pressure probably due to renal dysfunction due to alteration in the renal blood supply.

Mortality Rate

Mortality of 0% was observed in GRH group.

DISCUSSION

The importance of IAH and ACS are still not widely known. The present study (called GRH study hereafter) is an attempt to explore the incidence and importance of these conditions in the morbidity of surgical patients. Very few experimental studies have been made in these patients and since each person used different criteria, comparison is sometimes difficult.

While analysing the results, it was found that the incidence of increased intra-abdominal pressure (emergency) was more among men when compared to women. The number of females was more in the elective case list. Hence more males were suffering from intra abdominal hypertension. There was no significant difference between the two groups of increased and normal intra abdominal pressure with regard to mean age distribution.

Urea was significantly raised in the increased intra-abdominal pressure group when compared to the normal intra-abdominal pressure group at all time intervals (pre-operative, post – operative, 4^{th} hour, 12^{th} hour and 24^{th} hour), thus indicating renal dysfunction in cases of increased intra abdominal pressure. Further, the intra abdominal pressure and urea of each of these patients were monitored serially at regular intervals. There was no correlation between the two. This reinforces the concept that the duration of IAH is more important than the actual pressure. Similarly a significant increase in the creatinine level in all cases of increased intra-abdominal pressure was found when compared to

the normal group, at all time intervals. Similarly when monitored serially there was no correlation between the increased intra abdominal pressure and creatinine. Hence it reinforces the concept that, the duration of increased intra abdominal pressure is more significant than the actual pressure probably due to renal dysfunction due to alteration in the renal blood supply.

There was no significant correlation between the urine output and the intra-abdominal pressure. This might indicate that urine output might not be an ideal indicator of renal dysfunction in cases of intra abdominal hypertension or abdominal compartment syndrome.

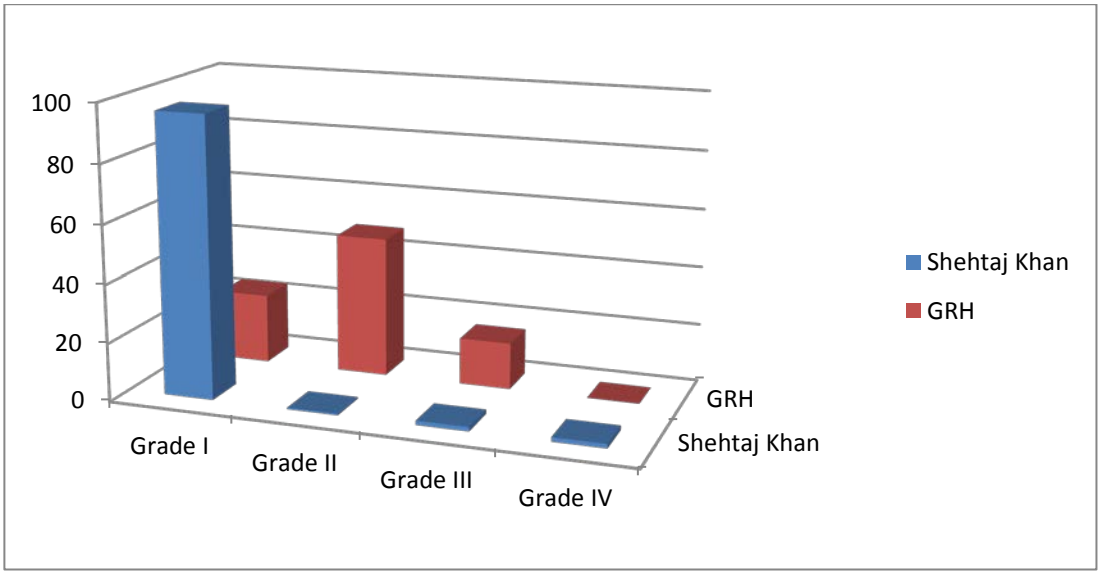
There was no significant difference in the incidence of atelectasis pre-operatively between the two groups which was tested clinically and with the help of chest X-Ray. There is a mild increase in the incidence of atelectasis post-operatively in cases of increased intra-abdominal pressure as compared to the group with normal intra abdominal pressure. This might indicate that pulmonary atelectasis is related to the duration of intra abdominal hypertension since the post operative intra abdominal pressure is more in the emergency cases than the elective cases.

Mechanical ventilation was not needed for both the groups post operatively.

Mortality Rate

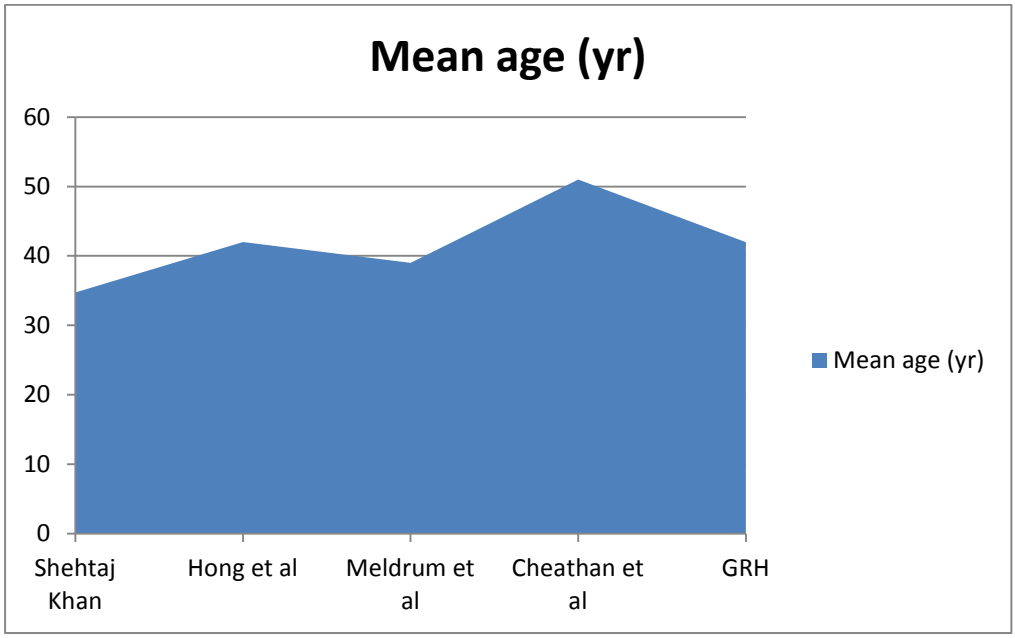
Mortality of 0% was observed in GRH group.

Fig 24: Comparison of the Grades of IAH between the studies



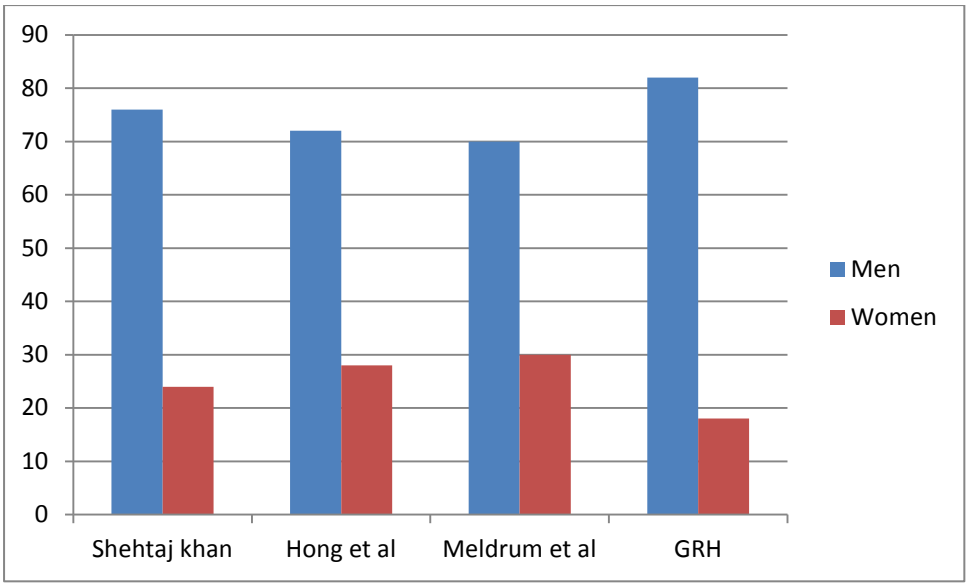
In the Shehtaj khan et al study most patients were in Grade I intra abdominal hypertension(96%), while in GRH most cases were prevalent in Grade II intra abdominal hypertension(48%). Also relatively more cases presented with Grade III intra abdominal pressure as compared to the other study which are 16% and 1.5% respectively.

Figure 25: Comparison of Mean age between the various case series



Regarding the age of incidence, the GRH study did not find any significant difference in the mean age group of elective and emergency patients. The large standard deviation indicates the large scatter around the mean. Other studies reported mean age groups varying between 30 and 50 yrs. This difference could be due to differences in experimental design.

Figure 26: Comparison of Gender distribution in Emergency laparotomy patients between the various case series



When the sex difference was analysed, incidence of the disease in men was seen to be significantly higher than in females in all studies. This could be due to the higher incidence in accidents in men.

RENAL DYSFUNCTION

Increase in the intra-abdominal pressure is believed to affect renal function especially renal filtration. The commonly performed glomerular function tests are blood urea and creatinine. A much more sensitive test is creatinine clearance but this test cannot be done properly in patients undergoing emergency surgery. GRH and other studies show a significant increase in blood urea and creatinine levels. GRH study did not find any significant difference in the urine output. The reasons for this could be that tubular function may not be affected by IAH and one more important factor viz., fluid balance is another influence which can affect urinary output.

A comparison of the renal dysfunction which has been calculated using the RIFLE'S criteria, revealed a renal dysfunction of 44% in GRH pre-operatively. On comparing it to other studies, Shehtaj Khan series had a high incidence of renal dysfunction upto 78% while Sugrue had 69%. The fall in the post operative creatinine as compared to pre operative creatinine was similar in all the three case series.

Figure 27: Comparison of the Pre and post operative creatinine values between the various studies

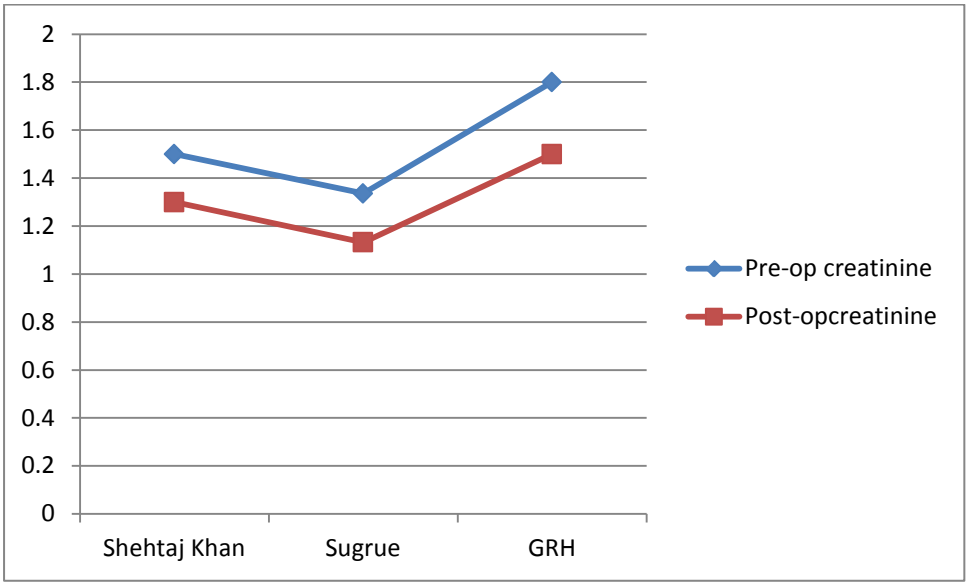
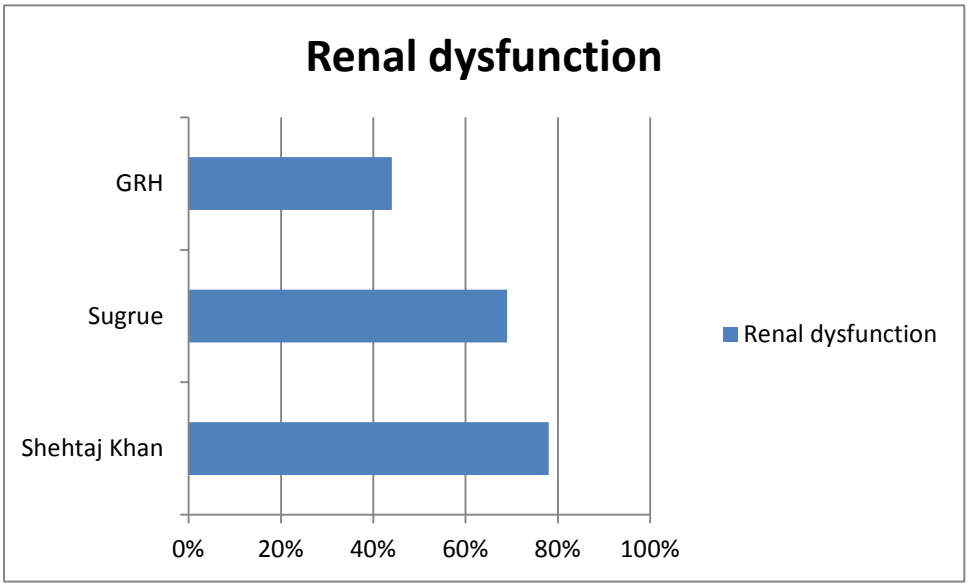


Figure 28: Comparison of the percentage of pre-operative renal dysfunction between the various case series



RENAL DYSFUNCTION COMPARISON

Table 13: Pre operative and post operative renal dysfunction – case distribution in Shehtaj Khan case series

Shehtaj Khan	Post-operative		
Pre-operative		**No Dysfunction**	**Dysfunction**
	Dysfunction	**15.8%**	**33.5%**
	No Dysfunction	**47.5%**	**3.2%**

Table 14: Pre operative and post operative renal dysfunction – case distribution in GRH case series

GRH	Post-operative		
Pre-operative		**No Dysfunction**	**Dysfunction**
	Dysfunction	**16%**	**30%**
	No Dysfunction	**54%**	**0**

The ratio of renal Dysfunction and non-dysfunction both pre and post operatively seem to be similar in both the studies. It was found to be 47.5% and 54% in the studies conducted by Shehtaj Khan and GRH respectively.

RESPIRATORY DYSFUNCTION

The increase in intra-abdominal pressure could affect pulmonary function due to the upward displacement of the diaphragm. The commonly expected pathology is atelectasis of the bases of lungs. Post operatively, a small percentage of patients with increased IAH had atelectasis although there was no difference pre - operatively. None of the groups needed the use of ventilator. The cases of IAH with Respiratory dysfunction, both pre and post operatively seem to be more prevalent in the Shehtaj Khan(74.7%) studies where as in GRH the patients with No-respiratory dysfunction pre and post operatively seem to be more prevalent(78%).

Table 15: Pre operative and post operative respiratory dysfunction – case distribution in Shehtaj Khan case series

Shehtaj Khan	**Post-operative**		
Pre-operative		**No Dysfunction**	**Dysfunction**
	Dysfunction	**13.9%**	**74.7%**
	No Dysfunction	**5.7%**	**5.7%**

Table 16: Pre operative and post operative respiratory dysfunction – case distribution in GRH case series

GRH	**Post-operative**		
Pre-operative		**No Dysfunction**	**Dysfunction**
	Dysfunction	**0**	**10%**
	No Dysfunction	**78%**	**14%**

The cases of IAH with Respiratory dysfunction, both pre and post operatively seem to be more prevalent in the Shehtaj Khan(74.7%) studies where as in GRH the patients with No-respiratory dysfunction pre and post operatively seem to be more prevalent(78%).

CONCLUSION

ACS/ IAH is associated with profound physiological abnormalities both outside and within the abdomen. While treating these patients it is essential to identify the signs of increased abdominal pressure early and start the management accordingly. It is also important to monitor the intra-abdominal pressure of the affected patients and those with (more than two) risk factors either continuously or intermittently. Understanding the pathophysiology of ACS/IAH is of prime importance for applying patient tailored treatment. If needed, appropriate surgical intervention should be done at the stage of IAH itself and should not be postponed till the development of ACS.

Renal dysfunction is the most common complication of abdominal compartment syndrome. Pre-operative renal dysfunction was found to be high in all the case series ranging from around 40% to 80%.The fall in the post operative creatinine as compared to pre operative creatinine was also observed in all the case series. In many of the studies renal dysfunction became evident as oliguria and later progressed to anuria. Compression of the renal vein and parenchyma and reduced renal perfusion, lead to reduced microcirculation to the functioning glomeruli and cortex. This results in tubular and glomerular dysfunction and substantially reduced urine output since FG = MAP – 2 x IAP

Thus the IAH induced renal dysfunction and prerenal azotemia will neither be responsive to fluid resuscitation nor vasopressors. It improves dramatically by appropriately and promptly reducing the elevated IAP.

The awareness of the entity called Intra abdominal hypertension and abdominal compartment syndrome is spreading in recent times. Yet in many of the centers it is still under-diagnosed as strict protocols to monitor intra abdominal pressure in critical care patients both in the medical and surgical side have not been laid down. As recommended by the World Society of Abdominal Compartment Syndrome, all cases in the critical care wards should be assed for intra abdominal pressure immediately following admission and serially in cases of elevated initial pressure. Though the mortality rate is zero in this case series, it is probably due to the choosing patients with increased abdominal pressure selectively rather than monitoring all the patients in the critical care ward, some of whom may have died because of undiagnosed Intra abdominal hypertension.

Hence abdominal compartment syndrome is a treatable condition when it is timely diagnosed and appropriately managed. Both medical and surgical treatments play equally important role in their management. Awareness and recognition of this entity will go a long way in reducing the mortality of many critically ill patients and all it takes is a simple bedside test to make the difference between probable death and survival.

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ABBREVIATIONS & ACRONYMS

ACS	:	Abdominal compartment syndrome
IAH	:	Intra-abdominal hypertension
CT	:	Computerised tomography
ICU	:	Intensive care unit
WSACS	:	World Society Abdominal Compartment Syndrome
APP	:	Abdominal perfusion pressure
MAP	:	Mean arterial pressure
IAP	:	Intra-abdominal pressure
FG	:	Filtration gradient
GFP	:	Glomerular filtration pressure
PTP	:	Proximal tubular pressure
ITP	:	Intra-thoracic pressure
PEEP	:	Positive end expiratory pressure
CVP	:	Central venous pressure
PAOP	:	Pulmonary artery occlusion pressure
ICP	:	Intra-cranial pressure
CSF	:	Cerebrospinal fluid
CPP	:	Cerebral perfusion pressure
VAC	:	Vacuum assisted closure
SD	:	Standard deviation

PROFORMA

Name: **Age/Sex:**

Diagnosis :

Procedure planned :

BMI:

Co-morbid illness :

On Examination

Pulse : **Blood Pressure :**

Respiratory Rate : **Saturation :**

Temperature :

S/E

CVS: **RS:**

Per Abdomen:

	Pre-op	Immediate	4hrs	8hrs	12hrs	16hrs	20hrs	24hrs
Urea								
Creatinine								
Urine Output								
Intra-abdominal Pressure								

Hb:

CXR:

Operative Findings:

Operative procedure

Relaprotomy : Yes / No -(Reason if yes)

Post –op ventilation :

Mortality (Cause) :

ETHICAL COMMITTEE APPROVAL

MASTER CHART

Name	Age (yrs)	Sex	Ip No	Diagnosis
Paneer Selvam	50	M	108425	Multiple stab-jejunum
Manjunathan	35	M	109141	Jejunal perforation
Janarthanan	65	M	109552	Obstructed umbilical hernia(jejunum)
Nagaraj	40	M	109702	stab injury- transverse colon injury
Raghavan	65	M	109239	obstructed inguinal henia
Asha	50	F	110230	Stab injury-jejunal perforation with peritonitis
Venkat	44	M	110634	Sigmoid Volvulous
Nagappan	64	M	111104	Ileal Perforation
Ramani	54	F	111370	Obstructed Incisional hernia
Shankar	63	M	111473	Obstructed umbilical hernia
Vijaya	40	F	111660	Ileal Perforation
Selvaraj	65	M	112010	Obstructed incisional hernia
Vasudevan	33	M	112115	Appendicular perforation
Manikam	50	M	112861	Obstructed umbilical hernia
Visaladevi	21	F	114814	Ileal Perforation
Jagadeesh	25	M	114939	Obstructed Inguinal hernia
Moya	25	M	113	Ileal Perforation
Iyappan	49	M	777	Duodenal perforation
Rajkumar	25	M	1964	Ileal Perforation
Dinesh	22	M	2101	Jejunal perforation
Rajesh	30	M	2064	Incisional hernia with jejunal gangrene
Amitha	18	F	2190	Ileal Perforation
Srinivasan	65	M	3314	Left obstructed inguinal hernia
Vinoth	19	M	4206	Ileal Perforation
Rani	60	F	4432	Obstructed Incisional hernia
Saravanan	40	M	4848	Ileal Perforation
Periaymani	45	M	5045	Obstructed paraumbilical hernia
Sahul	55	M	5169	Right obstructed inguinal hernia
Sebastian	22	M	5435	Multiple ileal perforation
Ramesh	59	M	5684	Gastric perforation
Micheal	22	M	5970	Ileal Perforation
Nagappan	60	M	5957	Obstructed Incisional hernia with small bowel g
Assama	21	M	7100	Duodenal perforation
Kalyani	20	F	7116	Obstructed Incisional hernia
Rajavel	35	M	7128	Strangulated left inguinal hernia
Selvakumar	22	M	7368	Obstructed left inguinal hernia
Arivalagan	52	M	8244	Ileal Perforation
Rambayadan	24	M	9787	Ileal Perforation
Rajendran	40	M	9852	Stomach perforation
Sundaramoorthy	40	M	10145	Duodenal perforation
Anandraj	48	M	10249	Blunt injury abdomen-jejunal perforation
Shenbagavalli	56	F	10563	Left obstructed inguinal hernia with ileal gangre
Ayesha	60	F	10663	Obstructed incisional hernia
Saravanan	25	M	11534	Ileal Perforation
Dhanasekhar	41	M	13208	Duodenal perforation

Narayanammal	52	M	14382	Ileal Perforation
Babu	37	M	15225	Duodenal perforation
Yasin	51	M	15512	Jejunal perforation
Gunasekar	50	M	16207	Gastric perforation
Papitha	45	M	21743	Gastric perforation

Ponnavel	56	F	11061	carcinoma stomach
Devi	29	F	998630	Calculous cholecystitis
Shanthi	46	F	101255	Cholilithiasis
Thillaikrishnan	66	M	100729	Gastric outlet obstruction
Shakthivel	32	F	103052	GB polyp
Latha	39	F	103978	Ventral hernia
Harikrishnan	13	M	104792	Rectal prolapse
Padmashree	36	F	105719	Incisional hernia
Govindan	48	M	105719	Rectal prolapse
Durairaj	40	M	105746	Carcinoma ascending colon
Malliga	50	F	104187	Choledocholithiasis
Lokeshwari	34	F	106706	Cholelithiasis
Rathinammal	56	F	107937	Spigellian Hernia
Sulthana	52	F	10811	Cholelithiasis
Kamini	30	F	109311	Cholilithiasis
Mala	56	F	109364	Incisional hernia
Rajeshwari	35	F	109662	Hepatic flexure growth
Maariyamal	60	F	111394	Gastric outlet obstruction
Potkodi	55	F	111901	Incisional hernia
Shanthi	50	F	111381	Carcinoma splenic flexure
Annamalai	51	F	112339	Gastric outlet obstruction
Venkat	44	M	114742	Post sigmoid colectomy-paul mickulicz
Shanmugam	48	M	1274	Incisional hernia
Vijaya	40	F	2083	Status-end ileiostomy
Selvaraj	65	M	2160	Status-end ileiostomy
Shalini	45	F	2435	Paraumbilical hernia
Karthavarayan	86	M	2941	Gastric outlet obstruction
Chakarabani	58	M	4916	Carcinoma stomach with liver secondaries
Visalam	25	F	4560	Post end ileostomy status
Rajesh Krishnan	41	M	5958	Umbilical Hernia
Rajamani	45	M	8452	Gastric outlet obstruction
Syed Ali	19	M	11250	Carcinoma stomach
Pachaiyammal	60	F	12267	Carcinoma stomach
Shenbagavalli	56	F	13460	Carcinoma stomach
Shenbagavalli	50	F	10563	Post end ileostomy status
Kaja Mohideen	63	M	15004	Carcinoma stomach
Unnamalai	42	F	16213	Carcinama stomach
Rangan	71	M	15925	Carcinoma stomach
Venkatraman	57	M	16428	Carcinoma stomach
Joshwa	62	M	51991	Carcinoma eosaphagus

Subramani	43	M	52343	carcinoma stomach
Durai	60	M	8341	Post Cholecystectomy - choledochojejunostomy
Kamatchi	32	F	113920	Incisional hernia
Krishnaveni	42	F	114355	Ventral hernia
Thanikachalam	37	M	615	Cholelithiasis
Selvaraj	40	M	1064	Carcinamo stomach
Pushpalatha	35	F	21456	Ventral hernia
Thiagu Nisha	39	F	21435	Incisional hernia
Sri devi	35	F	3214	Carcinoma stomach
Ramanan	37	M	21655	Incisional hernia

Procedure	BMI	Intra-abdominal pressure (mmHg)								
		Pre-op	Post-op	4hrs	8hrs	12hrs	16hs	20hrs	24hrs	Pre-op
Resection and anastemosis	25.9	12	15	16	15	15	16	17	17	64
perforation closure	19.9	20	21	20	21	20	20	20	21	54
Meshplasty	24.6	25	22	23	23	23	24	23	23	70
Primary closure with ileostomy	23	13	9	9	10	10	10	9	9	50
Hernioraphy	25.9	18	15	14	14	15	14	14	14	54
Resection and anastemosis	24.1	12	10	10	9	9	9	9	9	72
Left Hemicoloectomy	22.8	24	18	19	18	18	19	17	19	56
Resection and anastemosis	22.1	17	21	17	19	19	19	14	14	56
Meshplasty	23.7	24	23	23	24	23	23	23	25	64
Meshplasty	26.9	22	20	22	22	22	23	23	23	48
End iliostomy	22.4	19	18	17	17	18	20	20	20	48
Anatomical closure	25.1	21	20	20	19	20	21	21	21	28
Appendicectomy	18.9	16	15	15	16	16	16	17	17	57
Meshplasty	20.6	19	17	17	17	18	17	18	18	48
Ileal perforation closure with loop	29.4	10	7	8	8	9	8	9	9	38
Hernioplasty	20.4	15	13	14	14	14	15	15	16	49
Ileal perforation closure	24	18	16	17	18	17	17	18	18	56
Omental patch repair	24.6	17	13	15	14	14	15	15	15	40
Ileal perforation closure	20.4	16	10	12	13	14	13	14	14	56
Jejunal perforation closure	19.6	21	17	18	19	18	17	19	19	35
Resection and anastemosis	26.8	12	10	9	9	9	8	9	9	32
Ileal Perforation closure	23.9	18	15	17	16	17	17	17	18	28
Left herniorhaphy	26.9	20	15	15	16	16	17	17	17	36
Ileal perforation closure	19.8	10	8	9	8	8	8	8	9	54
Meshplasty	20.6	19	20	21	21	21	22	21	22	58
Ileal Perforation closure	27.3	21	20	19	18	19	18	19	19	54
Meshplasty	28.5	20	22	22	21	22	23	23	23	36
Herniorhaphy	24.7	18	15	16	17	16	16	16	17	48
Ileal-resection anastemosis	23.9	19	17	17	17	17	17	17	17	54
Live Omental patch repair	21.8	12	14	14	14	14	14	15	15	38
Ileal perforation closure	27.4	14	14	14	15	15	16	15	15	42
Resection and anastemosis with h	24.7	18	19	20	19	19	20	21	21	68
Live Omental patch repair	20.7	12	10	9	10	10	11	11	11	34
Hernioplasty	24.6	19	20	20	20	20	19	19	20	48
Resectional anastemosis with left	25	16	12	14	13	14	14	14	15	36
Left herniorhaphy	22.7	17	14	13	14	15	15	14	15	30
Ileal perforation closure	27.5	17	12	13	14	14	14	14	14	40
Resection Anastemosis	29.5	19	17	16	16	16	16	17	16	64
Live Omental patch repair	20.5	13	10	10	10	10	11	10	10	58
Live Omental patch repair	19.7	14	12	12	12	13	12	12	14	38
Resection Anastemosis	19.6	23	15	17	16	16	16	16	17	40
Resection Anastemosis	24.7	17	13	14	13	14	15	15	16	40
Meshplasty	28.9	19	13	13	14	13	15	15	15	38
Ileal Perforation closure	23.5	10	5	6	7	7	6	6	7	36
Live Omental patch repair	20.6	13	7	9	8	8	8	8	9	52

Ileal Perforation closure	28.5	18	13	14	13	13	13	14	14	48
Live Omental patch repair	21.7	12	9	10	10	10	11	10	10	54
Resection Anastemosis	22.7	14	9	9	8	9	9	10	10	58
Live Omental patch repair	19.8	10	7	8	8	8	8	8	9	48
Live Omental patch repair	20.5	13	8	9	7	7	7	8	8	38
Feeding jejunostomy	17.9	1	8	8	8	8	9	9	9	28
open cholecystectomy	26.8	3	6	6	7	9	9	8	9	36
open cholecystectomy	28.9	2	9	10	10	10	9	10	10	42
TVGJ	19.5	1	7	8	6	8	8	7	8	38
open cholecystectomy	27.4	3	8	8	8	8	8	9	9	46
Meshplasty	29.6	5	11	12	12	12	13	13	12	44
Thiersh wiring	24.6	4	10	9	11	11	12	11	12	40
Meshplasty	29.6	5	13	12	15	15	15	15	16	24
Abdominal rectoplasty	26.4	3	13	12	13	13	13	14	14	30
Right extended hemicolectomy	19.7	7	9	10	12	11	11	11	12	40
open cholecystectomy with CBD e	27.9	4	8	9	8	8	8	9	10	32
Open cholecystectomy	28.3	5	9	9	9	10	11	11	12	28
Meshplasty	28.5	5	13	14	14	15	14	14	15	35
Open cholecystectomy	26.9	3	8	8	9	10	10	9	10	38
Open cholecystectomy	24.8	4	9	7	7	9	9	10	9	24
Meshplasty	29.7	6	14	14	13	14	14	14	15	38
Right extended hemicolectomy	23.8	5	10	9	9	9	12	11	12	36
TVGJ	20.3	3	7	7	7	7	9	8	9	24
Meshplasty	27.9	5	9	10	9	9	9	10	10	25
Left extended hemicolectomy	20.5	7	12	11	10	11	11	11	12	38
TVGJ	19.6	2	8	9	9	11	11	11	11	40
Colostomy closure	24.7	3	7	9	9	9	10	9	10	30
Meshplasty	28.5	6	12	11	12	12	13	13	13	28
ileostomy closure	24.7	4	7	8	7	8	8	10	10	24
ileostomy closure	23.8	4	9	10	9	9	9		10	28
Meshplasty	25.7	6	14	12	13	13	13	14	14	38
Gastrojejunostomy	21.8	3	7	9	8	8	7	9	9	39
Ant GJ	20.5	2	8	9	10	10	10	11	10	40
ileostomy closure	21.5	5	7	6	7	7	8	8	8	33
Meshplasty	24.9	7	12	12	13	13	14	13	13	24
TVGJ	20.4	3	7	9	8	7	8	8	8	40
Ant GJ	22.6	2	9	9	9	9	10	10	10	38
Feeding jejunostomy	18.9	3	9	9	9	7	8	9	9	40
Ant GJ	19.5	2	6	6	8	8	9	9	9	28
ileostomy closure	23.5	5	9	12	12	11	11	12	12	34
Ant GJ	20.1	3	10	10	10	11	11	12	12	40
Subtotal gastrectomy with billroth	21.4	2	8	8	8	9	8	10	10	34
Ant GJ	22	4	7	7	8	9	8	8	9	36
Subtotal gastrectomy with billroth	20.4	2	9	7	8	8	9	9	9	30
Feeding jejunostomy	19.7	3	8	9	9	9	9	9	9	28

Feeding jejunostomy	18.6	2	6	6	6	7	7	6	8	28
Feeding jejunostomy	29.6	10	10	11	11	12	11	12	12	36
Meshplasty	27.9	7	9	11	10	10	10	11	10	27
Meshplasty	24.9	5	11	9	9	9	10	10	10	30
Open cholecystectomy	25.4	6	8	9	9	8	10	9	9	34
Subtotal gastrectomy with billroth	21.8	3	11	11	11	12	12	11	11	40
Meshplasty	21.5	4	8	8	9	9	10	10	9	36
Meshplasty	29.3	9	15	14	14	14	16	16	16	30
Feeding jejunostomy	21.1	2	9	8	9	9	7	9	9	28
Meshplasty	28.9	10	14	15	15	16	16	15	16	30

Urea (mg/dl)				Creatinine (mg/dl)					Urine output (m			
post-op	4hrs	12hrs	24hrs	Pre-op	Post-op	4hrs	12hrs	24hrs	0-4hrs	4-8hrs	8-12hrs	12-16hrs
56	48	48	42	2.3	2.4	2.4	2.3	2	350	900	450	600
52	46	47	40	1.4	1.1	1.1	1	1.1	450	400	400	500
66	56	52	54	4.5	3.8	3.6	2.8	2.9	250	300	300	200
48	48	50	48	1.2	0.8	1.1	1.1	1	500	400	450	500
56	56	55	52	1.2	1.1	1	1.1	0.9	400	600	475	450
68	70	56	60	5.8	5.3	3.4	3.2	2.8	150	200	200	1000
50	54	52	52	1.3	1.4	1	1.1	1	400	450	275	350
56	54	50	52	1.8	1.6	1.7	1.3	1.4	575	400	500	550
60	62	59	60	4.5	4.4	4	4.1	2.9	400	450	300	350
40	42	43	40	1	0.9	1.1	1	0.9	600	550	400	375
40	44	38	38	1.1	1.2	1	1.1	1.2	400	250	300	350
27	32	32	34	0.9	0.8	1.1	1	0.9	475	300	275	450
54	55	56	56	3.4	3.1	3.2	2.8	2.9	150	125	150	200
48	44	37	38	1.2	1.1	1	1.1	1.2	350	300	375	275
36	32	38	38	1.1	0.9	0.8	1.1	1	375	350	300	400
40	42	38	36	1.6	1.2	1.3	1.2	1.1	200	125	225	300
48	38	38	32	1.9	1	0.9	1.1	1	225	300	350	400
32	30	28	30	0.9	0.8	1.1	1	0.9	400	350	275	275
57	48	52	54	4.8	2.6	2.9	2.8	2.7	100	125	150	200
38	38	28	32	1	0.9	1.1	1	1.2	350	375	300	350
28	28	34	32	0.9	0.9	1	1.1	0.8	400	200	450	250
30	28	34	32	1	0.7	0.8	1.1	0.9	300	350	250	225
34	30	38	29	1.1	1	0.9	1.1	1	225	400	250	275
56	50	44	48	2	1.9	1.7	1.5	1.3	275	125	175	150
50	40	44	44	3.1	3	3.2	3	2.8	150	100	150	200
47	42	44	42	1.2	1.1	1.2	1	1.1	250	150	150	175
34	36	34	32	1	0.8	1.3	1.4	1.1	275	250	200	300
44	42	42	44	1.2	1.1	1.2	1	0.9	250	300	350	300
48	46	40	44	1.7	1.5	1.6	1.4	1.4	275	300	150	175
30	38	40	38	0.8	0.8	0.9	1	0.8	300	175	250	200
40	40	42	40	1.2	1.1	1.2	0.9	1	200	200	250	150
62	66	70	64	4.2	4.1	4.3	4	4.1	150	200	200	150
40	36	34	34	1.1	1	0.8	1.1	1	200	125	250	175
44	42	44	38	2	1.1	0.9	0.8	0.9	250	200	150	200
32	36	36	30	1.1	0.9	1	0.8	0.9	200	250	125	150
28	34	32	32	1	0.9	0.8	1.1	1	200	150	175	150
38	32	36	34	1.2	1	1.1	1.1	1.2	225	150	175	200
56	62	60	58	4.6	4.5	4.6	5	4.5	100	50	150	50
48	50	52	62	1.7	1.5	1.4	1.5	1.4	150	100	125	150
40	38	38	40	0.9	1	0.9	1.2	1	200	150	175	150
38	34	30	38	1.2	1.1	1	1.1	0.8	225	300	275	150
42	40	38	42	1.1	0.9	0.8	1	0.9	225	250	175	175
40	40	36	34	1.2	0.9	1.1	1	0.9	175	200	150	125
34	40	40	38	1	1.1	0.9	0.9	1.2	225	250	175	225
54	50	50	48	2.7	1.5	1.4	1.5	1.6	200	225	175	250

44	46	38	36	1.2	1.3	1.1	1	1	0.8	150	225	300
56	55	50	54	1.5	1	1.3	1.4	1.2	175	175	150	175
54	60	54	48	3.8	3.6	4	3.2	3.3	150	100	100	50
46	46	40	48	1.2	1	1.3	1.1	0.9	150	150	175	250
30	36	34	34	1.1	1	0.9	0.9	1.1	200	175	150	150
30	32	28	30	0.9	0.8	1	1.2	1	325	225	250	275
30	34	34	32	1.2	0.8	1	1	0.9	350	225	275	250
34	36	38	34	1.2	0.9	0.7	1.1	1	250	200	100	150
28	34	32	32	1.1	1	0.8	1	1.2	150	175	200	225
38	38	40	36	1.4	1.5	1.6	1.6	1.4	175	175	200	150
40	42	40	38	1.3	1.4	1.6	1	0.7	225	150	300	100
38	36	36	38	1.2	1.1	1.1	0.9	0.9	325	200	175	25
28	30	26	28	0.6	0.5	0.8	0.8	0.6	225	200	150	150
32	30	26	34	1.1	0.9	1	1	0.8	200	450	350	175
40	36	36	38	1.6	1.8	2	2.1	2.1	200	350	275	150
30	34	32	30	1	0.8	1	1.1	0.9	175	150	125	150
28	30	28	33	0.9	0.8	0.8	1.1	1	350	400	200	375
32	28	34	34	1	1.2	1.2	0.9	0.9	175	150	225	350
36	32	34	34	1.2	1	0.7	0.9	0.9	225	175	400	175
28	28	30	26	1	1	0.9	1	1.2	350	200	375	175
28	34	34	28	1.2	1	0.9	0.9	1	275	200	375	500
40	42	40	28	2.1	2	2.1	1.9	1.7	300	275	400	275
28	24	30	28	1	1.2	1.1	1.1	0.9	600	175	175	425
28	26	30	28	0.9	0.8	1.1	1	0.9	200	200	350	125
32	30	28	26	1.1	1	1.2	1	0.9	150	175	125	200
36	34	36	37	1.2	1	0.9	0.9	1	225	400	150	150
28	28	28	30	0.5	0.7	0.8	1	0.8	250	100	200	175
27	24	30	28	1.1	1	1.2	1.2	0.9	175	300	150	175
28	25	28	30	0.9	0.7	1	1.1	1	200	175	275	225
24	28	30	28	0.7	0.6	1.2	1	1.2	225	250	175	350
36	34	37	32	1.2	1	1.1	1.1	0.9	600	150	250	175
40	42	40	40	1.1	1.2	1.2	1	0.9	250	325	400	100
30	34	32	32	0.9	0.8	1.1	0.8	0.8	275	200	175	350
28	34	30	28	0.9	0.9	1	1.1	1	200	250	250	200
26	24	24	28	0.5	0.7	0.7	0.6	0.7	175	200	225	175
38	42	36	34	1.2	1	0.9	1.1	0.9	200	175	325	300
36	38	37	40	1	1	0.8	1	1	250	200	300	275
42	40	38	36	1.3	1.4	1.2	1.1	1.1	325	300	250	275
30	30	28	34	1	1.1	1	0.9	0.8	450	300	275	250
34	36	40	37	0.9	0.9	1	0.8	0.7	375	400	250	250
38	36	37	34	1.2	1	1.1	0.9	0.9	600	250	325	400
36	40	38	37	1.1	0.9	1.2	1.1	1	400	300	325	200
38	36	30	32	1.1	0.8	0.9	0.9	1.1	450	250	200	375
30	27	28	30	1	1	1.2	0.9	0.7	450	300	600	300
30	28	34	32	0.7	0.6	1	0.9	1	650	300	300	250

30	32	32	38	0.8	0.9	1	1.1	0.9	375	250	325	275
34	33	40	36	1.1	1	1	0.9	1.1	400	550	300	325
30	28	30	32	0.7	0.9	1	1	1.2	300	350	375	300
29	30	34	34	1	0.9	1.2	1	1.1	450	300	250	325
32	38	33	30	0.9	0.7	1.1	1	1	300	350	450	400
44	42	40	38	1.4	1.1	1.2	1	1	275	300	250	250
34	37	40	38	1	0.9	0.8	1.1	1	300	450	350	300
28	32	32	35	1.1	1	0.9	1	1	300	325	275	200
30	31	33	30	0.9	0.8	1	1	1.1	275	200	250	250
28	29	33	32	1.2	1	0.9	1	1.1	250	250	275	200

ıl)		Atelectasis		Post-op ventillation	Re-laparotomy
16-20hrs	20-24hrs	Pre-op	Post-op	Yes/No	Yes/No
400	400	No	Yes	No	No
550	600	No	No	No	No
400	300	No	No	No	No
250	600	No	No	No	No
500	400	No	No	No	No
600	200	No	Yes	No	No
200	450	No	No	Yes	No
400	150	Yes	Yes	No	No
400	450	No	No	No	No
400	500	No	No	No	No
400	450	No	No	No	No
275	350	No	No	No	No
200	150	No	Yes	No	No
275	350	No	Yes	No	No
575	200	No	No	No	No
250	300	No	No	No	No
300	350	No	No	No	No
375	450	No	No	No	No
100	150	Yes	Yes	No	No
250	150	No	No	No	No
275	350	No	No	Yes	No
300	350	No	No	No	No
300	250	No	No	No	No
125	300	No	No	No	No
250	200	No	Yes	No	No
250	150	No	No	No	No
150	200	No	No	No	No
350	200	No	No	No	No
200	225	No	Yes	Yes	Yes
150	200	No	No	No	No
175	250	No	No	No	No
300	175	No	Yes	Yes	No
250	250	No	No	No	No
250	200	No	No	No	No
225	250	No	No	No	No
250	200	No	No	No	No
225	200	No	No	No	No
100	150	Yes	Yes	No	No
175	150	No	No	No	No
225	200	No	No	No	No
200	300	No	No	No	No
150	200	No	No	No	No
250	150	No	No	No	No
200	150	No	No	No	No
125	200	No	Yes	No	No

175	200	No	No	No	No
200	225	No	No	No	No
150	100	No	Yes	No	No
225	175	No	No	No	No
175	200	No	No	No	No
250	250	No	No	No	No
325	250	No	No	No	No
175	150	No	No	No	No
200	175	No	No	No	No
175	250	No	No	No	No
150	175	No	No	No	No
125	150	No	No	No	No
375	225	No	Yes	No	No
225	350	No	No	No	No
550	100	No	No	No	No
175	200	No	No	No	No
175	250	No	No	No	No
175	400	No	No	No	No
175	200	No	No	No	No
400	225	No	No	No	No
200	375	No	No	No	No
300	350	No	No	No	No
200	225	No	No	No	No
150	200	No	No	No	No
350	175	No	No	Yes	No
100	325	No	No	No	No
225	175	No	No	No	No
400	225	No	No	No	No
400	175	No	No	No	No
500	175	No	No	No	No
200	225	No	No	No	No
275	150	No	No	No	No
200	325	No	No	No	No
400	175	No	No	No	No
175	200	No	No	No	No
325	450	No	No	No	No
400	450	No	No	No	No
300	175	No	No	No	No
200	300	No	No	No	No
300	250	No	No	No	No
300	225	No	No	No	No
350	350	No	Yes	Yes	No
200	300	No	No	No	No
325	275	No	Yes	No	No
275	375	No	No	No	No

250	200	No	No	No	No
300	35	No	No	No	No
250	250	No	Yes	No	No
175	400	No	No	No	No
275	200	No	No	No	No
175	200	No	No	Yes	No
275	300	No	No	No	No
250	275	No	No	No	No
300	250	No	No	No	No
225	300	No	No	No	No

KEY

IAP (mmHg)
Grade I 12-15
Grade II 15-20
Grade III 20-25
Grade IV >25

UREA (n): 20-40mg/dl
Creatinine(n):0.7-1.2mg/dl
Urine output(n): >1ml/kg/hr